

THE OCCURRENCE AND SIGNIFICANCE OF BENIGN
PROTEINURIA IN YOUNG MEN IN CIVIL LIFE
WITH SPECIAL REFERENCE TO ITS INFLUENCE
ON PHYSICAL EFFICIENCY.

by

ARTHUR J. WILSON, M.B., Ch.B.

Thesis submitted for the Degree of M.D.

March, 1928.



TABLE OF CONTENTS.

	<u>Page.</u>
I. HISTORICAL INTRODUCTION	1-6
II. METHOD OF INVESTIGATION AND DETAILED CASE RECORDS.	7-63
III. ANALYSIS OF FREQUENCY OF BENIGN PROTEINURIA AND AGE-PERIOD INCIDENCE.	65-71
IV. TYPES OF PROTEIN PRESENT AND OTHER URINARY CHARACTERISTICS.	73-80
V. THE ORTHOSTATIC REACTION AND CYCLICAL PROTEINURIA.	81-83
VI. PHYSICAL CONSTITUTION AND HABITUS OF CASES SHOWING THE CONDITION.	84-85
VII. EFFECT OF MUSCULAR EXERTION ON THE PROTEINURIA,	86-90
VIII. EFFECT OF FEBRILE INFECTIONS AND INTERCURRENT DISEASES.	91-96
IX. BLOOD PRESSURE OBSERVATIONS.	97-99
X. INFLUENCE OF BENIGN PROTEINURIA ON PHYSICAL EFFICIENCY AND HEALTH RECORD.	100-103
XI. CONCLUSIONS.	104-105
XII. REFERENCES.	106-108
XIII APPENDIX (Details of Control Cases).	109-121

FOREWORD.

In carrying out the observations embodied in the ensuing thesis the writer has endeavoured, in a very modest way, to apply the teaching of the late Sir James Mackenzie - that general practice offers a unique field of certain types of medical research.

For this purpose he is fortunately situated for clinical material, being a member of a large medical practice of five doctors which has eight thousand National Insurance Act patients alone attached to it. Further, the age and sex distribution of the population of Coventry is most favourable to this research as the motor car and cycle industry attracts to it a large number of young males from other districts.

The work was much facilitated by the system of record-cards used in the National Health Insurance organisation, more especially in the compilation of the sickness statistics over a period of years.

Finally the writer is indebted to his partners for their kindness in referring to him some of the clinical material at the required age-periods.

HISTORICAL INTRODUCTION.

The significance of albumin in the urine of apparently healthy young people has been the subject of much discussion since Ultzmann¹ in 1870 suggested the possibility of this urinary finding without an accompanying nephritis.

Previously, following Bright's discovery² in 1827 of the association of albumin in the urine and nephritis, albuminuria was regarded as definite evidence of renal disease. Continuing Ultzmann's work, Leube³ in 1877 proved that albumin is found in the urine of many apparently healthy young people.

Work on this question was then eagerly taken up, especially in Great Britain, by Clement Dukes⁴ and Grainger Stewart and Gulland.⁵ Medical officers of schools had opportunities of examining large numbers of youths in their teens, and of following them during their years of school training and in many instances e.g. in Dukes's Rugby scholars, of watching them for a period of several years afterwards. In all cases it was found that as the youth matured, the albuminuria tended to clear up and that there was nothing to prognosticate any variation from future good health (especially from the point of view of subsequent nephritis) as compared with boys who had not shown this/

this urinary condition.

The question of the development of organic nephritis later in life in these cases of youthful albuminuria has been studied also by insurance medical officers. Fox⁶ was able to observe 20 consecutive cases showing the condition, for a period of over 30 years and he found that no evidence appeared from the after history, of any subsequent renal trouble. Saundby⁷ recorded 24 cases, over a period ranging from 6 to 13 years, in which none had passed on into Bright's disease. Goodhart⁸ noted the after history of 39 cases with a like experience, though the period of observation must be considered short (5 years).

American insurance experience, however, does not entirely agree with these findings. Dublin⁹ studied the after history, during a period of 10 years, of 5,000 persons who were rejected by the Metropolitan Life Insurance Co. of New York because of occasional albuminuria, even in the slightest possible trace. He found that, compared with the mortality figure for the general population (Glover's tables), those for the cases under consideration were 114% of the expected deaths. While compared with the mortality tables on American Business Men (1918) which reflects the actual experience among the best types of insured lives in recent years among American companies, the total mortality was not far from double. The excessive mortality/

mortality was manifested at virtually every age period. Thus at the age period 15-24 years, it was 145% of the expected according to the American Men table. While at ages 35-44 the actual deaths were 267% of the expected according to the American Men table or nearly 3 times as high as the expected. The excess mortality varied somewhat according to the age of the person and to the amount of albumin and was primarily from an increase in the death rate from Bright's disease, from tuberculosis and organic heart disease. He concluded that the seriousness of albuminuria as a factor in mortality is established.

Barrenger and Warren¹⁰ studied the after histories of 396 men, who had been passed for life assurance, although at their examination they had shown albumin in the urine (without casts). They found that the mortality amongst these people is higher than among normal subjects, but that it was exceptional for it to be a symptom of incipient nephritis; there was a predisposition to tuberculous infection and on the whole they regarded this type of albuminuria as evidence of generally lowered resistance.

During the war opportunity was taken of an investigation, on a large scale, into the incidence of the condition amongst the recruits of the British draft. These were young men, passed as fit for military/

military service and presumably typical of the general youth of the country. It was, however, not possible to follow up such a large series of cases as to subsequent health during the remainder of their army life and to determine to what extent the albuminuria exerted any influence on the after medical history (apart from the question of Trench Nephritis to which reference will be made later).

Also the reports from the school medical officers mentioned above, give little information as to whether their albuminuric boys showed any difference in their general health and vigour during their school life as compared with the non-albuminurics. What had been chiefly sought after was to discover whether such albuminuria of the youthful variety, unassociated with the ordinary signs of nephritis, did or did not, in later life show development into definite renal disease.

On going through the literature, it is striking that little reference has been made to this question, viz. whether such benign albuminuria in youth has any influence on morbidity rate, i.e. in predisposition or otherwise, to attack by the ordinary major and minor illnesses and diseases existing under ordinary civil conditions.

It/

It seemed that observation on this point could be undertaken most easily by the family physician in private practice, for it is he who has most opportunities of regular and continuous observations of his cases, and who is freely called in for advice on the slightest deviation from health.

METHOD OF INVESTIGATION.

The plan adopted was to examine a consecutive series of youths and young men to discover the incidence of the condition; to keep those cases showing it under regular observation during a period of some years; to study its development in those cases during that time; to observe, during the period, the associated general health and efficiency, as compared with an equal number of the cases examined, in whom no albumin had been found.

The investigation has been confined to males only, because of the difficulty of satisfactory urinary examination in females under the conditions of private practice.

Beginning observations in 1923, every youth and young man coming into the consulting room was asked to pass a specimen of urine at the time. A large number were patients of the practice who came for examinations at the writer's request. If protein was found, a note was taken of the youth's name, age, occupation, previous medical history, and present physical condition, and he was kept under continuous observation, reporting at regular intervals, whether he was well or not, up to the end of the observation period (1927).

In this way, examination was made of 1250 young males, /

males, between the ages of 15-25 years, all unselected, and comprising all social, physical and economic types, the majority being young men of the artisan middle-class engaged in motor engineering work.

In all instances, extraneous causes of protein were excluded, e.g. urethral discharges, seminal emissions, and surgical conditions; and cases with a history of previous Scarlet fever or Diphtheria were rejected for the purposes of the present investigation.

Complete systematic examination of each positive case was carried out, with special attention to clinical signs of renal or cardio-renal disease, e.g. signs of oedema, increased blood-pressure, cardiac enlargement, arterial thickening, changes in fundus oculi. Finally an elaboration of the urinary examination, to be referred to later, was done.

Doubtful cases were further examined by special accessory renal tests, those employed being Maclean's urea concentration test, microscopical examination for casts,* "fixation of specific gravity" test, estimation of diurnal-nocturnal ratio, examination of the early morning specimen after the night's rest, and the effect of rest in bed upon the proteinuria.

* Lack of variation over more than a few points, of the specific gravity of the various specimens of urine passed during the 24 hour period.

CASE DATA.

The following are the detailed medical case histories, from the commencement to the end of the period of observation, of all cases showing protein in the urine.

S.R. --- means the "sickness record" in the number of days absent from work during that year. (Surgical causes or accidents are excluded).

U. ---- means urinary examinations, the numeral following being the number of urinalyses made during that year, but not necessarily during the time of absence by sickness, unless so stated.

a. --- means albumin.

g. --- means globulin.

pos... means urinalysis positive for protein.

neg... means urinalysis negative for protein.

The time of day the examination was made is indicated thus:- m --- morning (9-11 a.m.) aft.--- afternoon (2-4 p.m.) even'g.--- evening (6-8 p.m.).

"Early morning specimen" means that passed on first arising in the morning after the night's rest.

Cases in which no "complaint" is mentioned are cases who came for examination at the writer's request.

CASE 1./

CASE 1. aged 16. School-boy. Came for examination for admission to a Friendly Society. Previous health good except for Lobar Pneumonia (aged 10). No other infectious disease history. Development and posture, good. Not nervous. Never faints. Good colour. Ht. 5 ft. 7". Weight 9 st.

Urine. (aft.) S.G. 1014. Albumin only (trace). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{75}$ Recumbent $\frac{115}{75}$.

1923. S.R. 7 days Gastro-Enteritis. U. 2. both neg.
 1924. S.R. 0. U. 6. 3 pos. (1 m. 2 even'g).
 1925. S.R. 0. U. 6. 1 pos. (aft.)
 1926. S.R. 0. U. 7. 3 pos. (2 m. 1 aft.) No changes in cardio-vascular system.
 1927. S.R. 10 days Quinsy - during which (febrile) passed albumin and globulin (trace only).
 2 days later (fever gone) neg. U. 4. 1 pos. (aft.)

B.P. $\frac{125}{75}$. Arteries soft. No cardiac enlargement. No casts. Feels very well.

CASE 2. aged 23. Motor-body builder. Previous health good. No infectious disease history. Development and posture, good. No lordosis. Not nervous. Good colour. No faints. Ht. 6 ft. 2". Wt. 12 st. 12 lbs.

Urine (even'g) S.G. 1010. Albumin only (small amt.). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{75}$ Erect. Recumbent $\frac{120}{75}$.

1923. S.R. 10 days Gastro-Enteritis. U. 2. both neg.
 1924. S.R. 0. U. 5. 2 pos. (1 afn. 1 even'g).
 1925. S.R. 3 days Coryza. U. 5. all neg.
 1927. S.R. 0. U. 6 - all neg.

CASE 3./

CASE 3. aged 20. Motor mechanic. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Good colour. Faints 0. Ht. 5 ft. 10". Wt. 10 st. 12 lbs.

Urine (m). S.G. 1008. Albumin only (trace). No casts or cells. Many oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{80}$ Erect. Recumbent $\frac{120}{80}$

1923. S.R. 0. U. 2. 1 pos. (m.) after night work.
 1924. S.R. 10 days Rheumatism (non-febrile).
 U. 6. 2 pos. (even'g).
 1925. S.R. 0. U. 6. 1 pos. (aft.)
 1926. S.R. 10 days feverish coryza during which passed no protein. U. 5. 1 pos. (even'g).
 B.P. unaltered. No cardiac enlargement.
 1927. S.R. 0. U. 6. 2 pos. (even'g) B.P. $\frac{130}{80}$.
 No arterial thickening. No casts.
 No cardiac enlargement. Feels very fit.

CASE 4. aged 18. Motor-fitter. Came for examination for entrance into a Friendly Society. Previous health good. No infectious disease history. Development good. Posture - slight lordosis. Good colour. Not nervous. Faints 0. Ht. 5 ft. 8 in. Wt. 10 st. 3 lbs.

Urine (even'g). S.G. 1012. Albumin only, small amount. No casts, cells or oxalates. Early morning specimen free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{75}$ Recumbent $\frac{115}{75}$

1923. S.R. 10 days Bronchitis. U. 2 - 1 pos. (even'g)
 1924. S.R. 0. U. 6 - all neg.
 1925. S.R. 0. U. 7 - 2 pos. (1 aft. 1 even'g).
 1926. S.R. 14 days Furunculosis. U. 5 - all neg.
 1927. S.R. 13 days Enteritis. U. 6 - all neg.

CASE 5. /

CASE 5. aged 25. Mechanic. Complaint - dental neuralgia. Previous health very good. Has never been medically treated before. Strong, athletic young man. Scratch man in cycle-track racing. Ht. 5 ft. 10". Wt. 10 st. 12 lbs. Not nervous. Posture good. No lordosis. Never fainted.

Urine (even'g. specimen). Protein present. Albumin only (small amount). Casts 0. Cells 0. Oxalates 0. Early morning specimen - free. No oedema. No cardiac hypertrophy.

B.P. $\frac{135}{90}$ Erect. Recumbent $\frac{130}{90}$.

1923. S.R. 0. U. 2. - 2 positive (1 aft. 1 ev'g).
 1924. S.R. 0. U. 5 (3 on day of track races, see p. 89.) of remaining two, one pos. (even'g)
 1925. S.R. 15 days Influenza. U. 3. one pos. (m).
 1926. S.R. 0. U. 4. - 2 pos. (both even'g).
 No change in clinical condition in cardiovascular system.
 1927. S.R. 7 days Coryza. U. 2. - 1 pos. (aft.).

B.P. unaltered $\frac{135}{90}$. No cardiac enlargement.
 Arteries soft. No casts. Feels very well.

CASE 6. aged 17. Mechanic. Previous health - good. Pale, tall, rapidly-growing. Poor posture with a degree of lordosis. Fainted occasionally at school. No infectious disease history. Ht. 5 ft. 11". Wt. 10 st. 4 lbs. Rather excitable.

Urine - (even'g). S.G. 1016. Albumin ($\frac{1}{8}\%$) and globulin. Casts 0. Cells 0. Oxalates - a few. Early morning specimen free. No oedema. No cardiac enlargement. Heart excitable. Arteries soft.

B.P. $\frac{135}{80}$ Erect. Recumbent $\frac{140}{85}$

1923. S.R. 0. U. 2. 1 pos. (aft.) g. only.
 1924. S.R. 3 days Coryza. 10 days Tonsillitis, during febrile period passed no protein.
 1925. S.R. 13 days Influenza. 5 days Dyspepsia. U. 6 - all neg.
 1926. S.R. 21 days Catarrhal Jaundice. U. 5 all neg.
 1927. S.R. 0. U. 5 all neg.

CASE 7. /

CASE 7. aged 24. Plumber. Complaint - whitlow. Previous health - 2 attacks of Pneumonia in childhood. No infectious disease history. Athlete. Development - posture good. Not nervous. No faints. Colour good. Ht. 5 ft. 10 in. Wt. 10 st. 7 lbs.

Urine (aft.) S.G. 1010. Protein (albumin only). Casts 0. Cells 0. Oxalates, a few. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{90}$ Erect. $\frac{120}{90}$ Recumbent.

1923. S.R. 21 days Bronchitis. U. 3. 1 pos. (aft.)

1924. S.R. 7 days Acute Follicular Tonsillitis. During the pyrexial period passed both albumin and globulin (latter in excess). 2 days later protein free. U. 3 1 pos. (even'g.)

1925. S.R. 0. U. 5. 1 pos. (even'g.)

1926. S.R. 4 days Coryza. U. 4. 1 pos. (aft.) No change in physical signs in cardio-vascular system.

1927. S.R. 0. U. 3. 1 pos. (even'g). No cardiac enlargement.

B.P. $\frac{128}{95}$. Feels very fit. Arteries soft. No casts.

CASE 8. aged 23. Mechanic. Complaint - sprained ankle. Development good. Posture good. No lordosis. Colour good. Not nervous. Previous health good. No infectious disease history. No faints. Ht. 5 ft. 7 in. Wt. 11 st. 3 lbs.

Urine (even'g) Albumin $\frac{1}{2}\%$. No globulin. S.G. 1008. Casts 0. Cells 0. Oxalates 0. No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{125}{85}$ Recumbent $\frac{120}{85}$

1923. S.R. 3 days Coryza. 10 days Bronchitis. U. 2 both neg.

1924. S.R. 10 days Influenza. U. 4 all neg.

1925. S.R. 17 days Influenza. 3 days Coryza. U. 3 all neg.

1926. S.R. 0. U. 5 all neg.

1927. S.R. 3 days Coryza. 14 days Tonsillitis. U. 6 all neg.

CASE 9. aged 24. Motor mechanic. Complaint - swelling of legs. Previous health - never feels very well. No infectious disease. Has had 2-3 attacks of Tonsillitis. Ht. 5 ft. 3". Wt. 10 st. 2 lbs.

Urine. (evening). large amount of albumin (.4% Esbach) No globulin. S.G. 1025. Casts - epithelial and fatty. Also cells. No oxalates. Early morning specimen contained a small amount of protein. Pronounced oedema over shins and ankles. Slight cardiac enlargement. Arteries - somewhat thick for age.

B.P. $\frac{164}{100}$.

Concentrates urea at 1.4%. Effect of recumbency has no effect on the albuminuria.

This case was considered definitely organic renal disease and was not included in the present observations.

(This patient died of Lobar Pneumonia in 1925).

CASE 10. aged 16. School-boy. Complaint - "cold" (afebrile). Previous health, good. No infectious disease history. Development poor. Posture poor with some lordosis. Somewhat nervous. Good colour. No faints. Ht. 5 ft. 3". Wt. 7 st. 2 lbs.

Urine (m.) S.G. 1015. Albumin only (small amt). No casts, cells or oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft. B.P. Erect $\frac{110}{80}$ Recumbent $\frac{105}{80}$.

1923. S.R. 10 days feverish Coryza, during which passed globulin and trace albumin. 2 days later (afebrile) both absent.

1924. S.R. 0. U. 6 all neg.

1925. S.R. 10 days Influenza. U. 6 all neg.

1926. S.R. 0. U. 5. 1 pos. m. Clinical signs unaltered.

1927. S.R. 3 days Coryza. 5 days Coryza. U. 6 - 2 pos. (even'g.)

B.P. $\frac{120}{85}$. No cardiac enlargement. Arteries soft. No casts. Feels very very well. Grown into a fine young man.

CASE 11. aged 23. Slaterer. Complaint - Pruritus. Previous health - very good. No infectious disease history. Development good. Posture good. No lordosis. Colour good. Athletic - football. Not nervous. Never faints. Ht. 5 ft. 7". Wt. 10 st. 8 lbs.

Urine (aft.) albumin ($\frac{1}{8}\%$) and a little globulin. No casts. A few oxalates. No cells. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$ Recumbent $\frac{120}{80}$

1923. S.R. 10 days Sciatica. U. 2. 1 pos. (albumin only) in morn. (after night work). After 6 hours recumbency, neg.
 1924. S.R. 3 days Coryza. 7 days Influenza. U. 6 - all neg.
 1925. S.R. 0. U. 5 - all neg.
 1926. S.R. 12 days Influenza. U. 5 - all neg.
 1927. S.R. 0. U. 6. all neg.

CASE 12. aged 20. Motor fitter. Previous health good, except for Pneumonia, aged 12. Pale complexion. Muscularity good. Ht. 5 ft 8". Wt. 11 st. 3 lbs. Posture good. No lordosis. Never fainted. Not nervous.

Urine (aft.) S.G. 1020. Protein albumin only (haze). Casts 0. Cells 0. Oxalates 0. Early morning specimen, free.

No oedema. No cardiac enlargement.

Arteries soft. B.P. $\frac{125}{85}$ Erect. Recumbent $\frac{130}{85}$.

1923. S.R. 15 days. Catarrhal Jaundice. U. - 2. 1 pos. (even'g.)
 1924. Left the town. No further particulars available.

CASE 13. /

CASE 13. aged 18. Butcher's assistant. Complaint - cut hand. Strong, muscular youth. Ht. 5 ft. 8". Wt. 9 st. 10 lbs. Colour good. Never fainted. Posture good. No lordosis. Not nervous. Suffered a good deal from Bronchitis in childhood, but health for the past few years has been very good. No history of any previous infectious disease.

Urine (evening) S.G. 1015. Protein present, albumin only (about $\frac{1}{4}$ Esbach). Casts 0. Cells 0. Oxalate crystals - a few.

Early morn. spec. of urine - free from protein.

No oedema. No cardiac enlargement. Arteries soft.

Blood pressure

Erect $\frac{120}{80}$ mm. Recumbent $\frac{120}{80}$ mm.

1923. S.R. 5 days, Furunculosis. U. 2. 1 pos. (aft.)

1924. S.R. 0. U. 5. 2 pos. (both m.)

1925. S.R. 10. Tonsillitis. Passed protein, albumin and globulin, (globulin predominating) during 3 days of pyrexia. Absent from 2nd day of fall of temperature. U. 5. 1 pos. (aft.).

1926. S.R. 0. U. 3. all negative.

1927. S.R. 0. U. 4 all negative.

CASE 14. aged 25. Electrician. Previous health good except for Lobar Pneumonia at age 12. No infectious disease history. Development and posture, good. No lordosis. Not nervous. Never fainted. Colour - pale. Ht. 5 ft. 7". Wt. 11 st.

Urine (m.) S.G. 1010. Albumin only (haze). No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{80}$ Erect. Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 2 both pos. (m.).

1924. S.R. 10 days, Influenza. 3 days Coryza. U. 5 all neg.

1925. S.R. 3 days Tonsillitis. U. 5 all neg.

1926. S.R. 0. U. 5 - all neg.

1927. S.R. 4 days Coryza. 8 days Influenza. U. 6 - 3 pos. (m. 1. even'g 1).

B.P. $\frac{130}{80}$ No cardiac enlargement. No casts. Arteries soft. Feels very well.

CASE 15. aged 19. Motor-body Builder. Complaint - muscular sprain. Athlete. Ht. 5 ft. 10 ins. Wt. 10 st. 12 lbs. Colour good. Muscular development, very good. Posture good. No lordosis. Not nervous. Never fainted. Previous health - good. No infectious diseases.

Urine (morning). Protein - albumin ($\frac{1}{4}$) and Globulin (latter in excess, i.e. more positive in cold acetic acid test). Casts 0. Cells 0. Oxalates - a few. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{98}$ Erect. Recumbent $\frac{120}{95}$

1923. S.R. 70 days - Rheumatism (non-febrile). U. 2. 1 pos. (even'g), alb. and glob.
 1924. S.R. 0. Before a rugby match - urine contained globulin in fair amount. No albumin. Specimen passed at end of match - no protein. 2 hours later no protein.
 1925. S.R. 14 days Influenza. U. 3. 1 positive (a. only) in early morning (after night work). 6 hours later, after rest in bed, no protein.
 1926. S.R. 10 days Acute Tonsillitis. During febrile period passed no protein. U. 4. all neg.
 1927. S.R. 3 days Coryza, 10 days Coryza. U. 6 - 1 pos. (albumin only) even'g.

B.P. $\frac{125}{100}$ No cardiac enlargement.

Feels very well. Arteries soft. No casts.

CASE 16. aged 24. Shop-assistant. Complaint - cut-finger. Previous health good. No infectious disease history. Development and posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 11". Wt. 11 st. 1 lb.

Urine S.G. 1010. Albumin (haze). No globulin. No casts or cells. Many oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{85}$ Recumbent $\frac{125}{85}$

1923. S.R. 0. U. 2. 1 pos. (even'g).
 1924. /

1924. S.R. 4 days Coryza. U. 6 all neg.
 1925. S.R. 0. U. 7 - all neg.
 1926. S.R. 21 days Bronchitis. U. 6 - all neg.
 1927. S.R. 0. U. 6 all neg.

CASE 17. Railway Porter, aged 20. Previous health good. Athlete. No infectious disease history. Posture and muscularity good. No lordosis. Colour good. Not nervous. No faints. Height 5 ft. 7 ins. Wt. 10st. 8 lbs.

Urine (even'g). S.G. 1012. Protein = albumin only (haze). Casts 0. Cells 0. Oxalates 0. Early morning specimen free.
 No oedema. No cardiac enlargement. Arteries soft

B.P. $\frac{135}{80}$ Erect. Recumbent $\frac{130}{80}$

1923. S.R. 0. U. 2 tests. 1 pos.(aft.).
 1924. S.R. 0. Before a rugby match this year urine was free of protein. Immediately after, and 2 hours after, no protein passed. U. 3 all negative.
 1925. Killed in a motor cycle accident.

CASE 18. aged 18. Mechanic. Complaint - tiredness. Previous health good until Scarlet Fever 3 years ago. Had "kidney inflammation" with it. Pale. Posture good. No lordosis. Never fainted. Ht. 5 ft. 8". Wt. 10 st. 6 lbs. Not nervous.

Urine (morning) S.G. 1020. Urine - much albumin (.3% Esbach). No globulin. Casts - numerous epithelial. Cells - a few red blood corpuscles. Oxalates 0. Early morning specimen - free.
 Some oedema of eyes. No cardiac enlargement.
 No arterial thickening.

B.P. $\frac{140}{90}$.

Concentrates urea at 1.7%. Urine passed after 3 hours rest in recumbent position still contained albumin.

D $\frac{4}{1}$ Total amount (daily) 35 oz.
 N. 1.

This was considered to be organic Nephritis and was not considered for the present investigation.

CASE 19. aged 16. Apprentice. Complaint - constipation. Previous health - always delicate. No infectious disease history. Ht. 5 ft. 4". Wt. 7 st. 10 lbs. Rather pale. Posture poor. Some lordosis. Nervous. Had faints in school.

Urine. (aft.) S.G. 1010. Protein = albumin and globulin (hazes only). Casts 0. Oxalates + + . Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{110}{82}$ Recumbent $\frac{120}{85}$

1923. S.R. 0. U. 2. 1 pos. (even'g).
 1924. S.R. 8 days Acute Tonsillitis. During febrile period passed albumin and globulin (former predominating). 2 days later a. still present. 4 days later absent. U. 3. (1 pos. aft.)
 1925. S.R. 0. U. 4 tests, all neg.
 1926. S.R. 10 days Influenza. U. 3. 1 pos. (even'g). No change in cardio-vascular system.
 1927. S.R. 0. U. 5. 2 pos. (both even'g.) no globulin.

B.P. $\frac{120}{90}$ No cardiac enlargement.
 Arteries soft. No casts.

Feels in good health.

CASE 20. aged 19. Carpenter. Strong athletic boy. Previous health good. No infectious disease history. Ht. 6 ft. 1". Wt. 11st. 2. Colour good. No faints. Posture good. No lordosis.

Urine (morning) S.G. 1015. Protein present in small amount. Albumin only. Casts 0. Cells 0. Oxalates 0. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{88}$ Recumbent $\frac{118}{85}$

1923. S.R. 0. U. 3. 1 pos. (even'g).
 1924. S.R. 0. U. 5. all neg.
 1925. S.R. 0. U. 4. all neg.
 1926. S.R. 14 days, Influenza. U. 4 all neg.
 1927. S.R. 0. U. 5. all neg.

CASE 21. aged 25. Complaint - Nausea. Had not felt well for past 18 months - dyspeptic symptoms. Previous health - fair. Numerous attacks of Quinsy.

Urine. S.G. 1020. Protein. 3% albumin. No globulin. Casts ++. epithelial and hyaline. Cells 0. No oxalates.

No oedema. No cardiac enlargement. Arteries soft. Examination of fundus, negative.

B.P. * $\frac{150}{100}$ Concentrates urea at 1.6%.

Protein absent from early morning specimen, but present after 6 hours rest in bed during the day. This case was considered Nephritic.

CASE 22. aged 15. School-boy. Complaint - cut head. Previous health - good. Operation appendicitis 2 years ago. No infectious disease history. Development good. No lordosis. Not nervous. Colour good. No faints. Ht. 5 ft. 4". Wt. 7 st. 2 lbs.

Urine (morning). S.G. 1010. Albumin (less than $\frac{1}{4}\%$) and globulin (trace). No casts or cells. A few oxalate crystals. Early morning specimen free. No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{110}{75}$ Recumbent $\frac{105}{75}$.

1923. S.R. 10 days Bronchitis. 3 days Coryza. U. 2 - both neg.

1924. S.R. 0. U. 5. all neg.

1925. S.R. 14 days Influenza. U. 6 - all neg.

1926. S.R. 6 days Gastro-Enteritis. U. 5 all neg.

1927. S.R. 10 days cold. U. 6 all neg.

CASE 23. aged 19. Labourer. Previous health good. No infectious disease history. Posture and muscularity good. Ht. 6 ft. Wt. 11 st. 8 lbs. Colour good. Not nervous. No lordosis. No faints.

Urine. (morning) Protein - albumin and globulin, haze only of each. Casts 0. Cells 0. Oxalates 0. Early/

Early morning specimen free.
No oedema. No cardiac hypertrophy.

B.P. Erect $\frac{125}{90}$ Recumbent $\frac{120}{85}$

1923. S.R. 0. U. 3 2 pos. (aft.). alb. only.
1924. S.R. 0. U. 2. both neg.
1925. S.R. 0. U. 4. 2 pos. (even'g.).
1926. S.R. 14 days Influenza. U. 4 all neg.
1927. S.R. 7 days Influenza. U. 2. both neg.

CASE 24. aged 19. Carpenter. General health good.
No infectious disease history. Development
average. Posture good. No lordosis.
No faints. Not nervous. Colour good.
Ht. 5 ft. 6". Wt. 10 st. 2 lbs.

Urine (aft.). S.G. 1015. Protein - albumin 0.
Cold Acetic acid test - strong positive reaction.
Casts 0. Cells 0. Oxalates 0. Early morning speci-
mens free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect. Recumbent $\frac{120}{80}$

1923. S.R. 0. U. 2. On both occasions, strong
globulin reaction. (A trace of albumin on
one occasion.)
1924. S.R. 0. U. 4. all positive (globulin only,
3; a. and g. 1). Effect of 6 hours rest in
recumbent posture - the protein disappeared.
1925. S.R. - 10 days - feverish cold. During febrile
period passed albumin and globulin (about
equally strong reactions). 2 days after cessa-
tion of fever - globulin only.
1926. S.R. 10 days Influenza. U. 4 - all pos. (glob.
only 2. alb. and glob. 2). No change in
cardio-vascular system. No casts.
1927. S.R. 8 days Tonsillitis. U. 5 all pos.
(m. 2. aft. 3). g. 4. a and g 1. No casts.
No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{85}$. Feels very well.

CASE 25./

CASE 25. (brother of last) aged 15. Message boy. General health good. No infectious disease history. Posture fair. No lordosis. Colour pale. Not nervous. No faints. Growing rapidly. Ht. 5 ft. 7". Wt. 8st. 5 lbs.

Urine (m.) S.G. 1020. Protein, - globulin only in fairly large amt. No casts, cells, or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{110}{70}$

Recumbent $\frac{110}{70}$.

1923. S.R. 17 days Bronchitis. U. 2 both pos. glob. only (1 aft. 1 even'g).

1924. S.R. 0. U. 5. 3 pos. (g. only) 1 m. 2 aft.

1925. S.R. 20 days Influenza. U. 4. 2 pos. glob. only, both m. Urine positive on one of the mornings, was negative 6 hrs. later after recumbency.

1926. S.R. Coryza 3 days. During febrile period passed albumin (haze) and globulin (predominating) 2 days after cessation of fever, g. (trace) only. No changes in cardio-vascular system. No casts.

1927. S.R. 0. U. 6. 3 pos. (all even'g). glob. only.

No casts. B.P. $\frac{120}{80}$. Arteries soft. No cardiac enlargement. Feels very well.

(Case 24 and 25 appear to fit in with Mackenzie Wallis's description of the condition of "Leaky Kidney", which according to him is a variety of functional proteinuria, unassociated with any evidence of organic disease, which tends to remain permanently and the urinary protein is largely globulin.).

CASE 26. aged 17. Apprentice. Previous health - good. No infectious disease history. Development and posture good. No lordosis. Good colour. Faints 0. Not nervous. Ht. 5 ft. 10". Wt. 10 st. 7 lbs.

Urine (even'g). S.G. 1012. Albumin only (a haze). No casts, cells or oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{85}$

Recumbent $\frac{128}{85}$

1923. S.R. 10 days Bronchitis. 4 days, Influenza.

U. 2. all neg.

1924. /

1924. S.R. 0. U. 5. 1 pos. morning, after night work. After 6 hours recumbency - neg.
 1925. Influenza 10 days. Feverish coryza 3 days. U. 5 - all neg.
 1926. S.R. 0. U. 7 - all neg.
 1927. S.R. 6 days, gastro-enteritis. U. 5 - all neg.

CASE 27. aged 15. School-boy. Complaint - Favus. Previous health good. No infectious disease history. Development fair. Posture rather poor. Some lordosis. Somewhat nervous. Good colour. Faints 0. Ht. 5 ft. 3". Wt. 7 st. 3 lbs.

Urine (aft.) S.G. 1014. Albumin ($\frac{1}{4}$) No globulin. No casts, cells or oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{110}{75}$ Erect. Recumbent $\frac{115}{75}$.

1923. S.R. 3 days Coryza. 5 days Diarrhoea. U. 2 - 1 pos. (aft.)
 1924. S.R. 0. U. 6 - all neg.
 1925. S.R. 0. U. 5 all neg.
 1926. S.R. 3 days Coryza. 5 days Dyspepsia. U. 6 - all neg.
 1927. S.R. 12 days Bronchitis. U. 5 all neg.

CASE 28. aged 24. School-teacher. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Not nervous. No faints. Good colour. Ht. 5 ft. 7". Wt. 10 st. 12 lbs.

Urine (m.) S.G. 1014. Albumin only (small amt.). No casts, cells or oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{80}$ Erect. Recumbent $\frac{125}{80}$.

1923. S.R. 3 days Coryza. 7 days Influenza. U. 2 - both neg.
 1924. S.R. 0. U. 5 - 2 pos. (even'g).
 1925. S.R. 12 days Influenza. 3 days Coryza. U. 6 - all neg.
 1926. S.R. 3 days Coryza. U. 5 all neg.
 1927. S.R. 14 days Coryza (febrile). U. 6 all neg.

CASE 29. aged 25. School-teacher. Complaint.-
Dyspeptic symptoms. Previous health very
good. No infectious disease history.
Build - very good. Colour good. Athletic.
Not nervous. No faints.

Urine (even'g). S.G. 1010. Albumin - trace only.
No globulin. Casts 0. Cells 0. Oxalates 0.
Early morning specimen free.

No oedema. No cardiac enlargement. No arterial
thickening.

B.P. $\frac{140}{95}$ Erect.

Recumbent $\frac{140}{95}$.

1923. S.R. 0. U. 2 tests 1 pos. (even'g).
1924. S.R. 6 days, feverish coryza. During febrile
period passed traces of albumin and globulin
(former in excess). 2 days after cessation of
temperature - protein free.
1925. S.R. 0. U. 5. 2 pos. Before a rugby foot-
ball match, urine was negative. 2 specimens,
one passed immediately after, the other 2 hours
after the match, both negative.
1926. S.R. 21 days Influenza. U. 4. 1 pos. (m.).-
specimen passed 6 hours later after recumbency
- negative. B.P. unaltered.
1927. S.R. 5 days Coryza. U. 4. all neg.

CASE 30. aged 19. Motor-mechanic. Previous health
good. No infectious disease history.
Development good. Posture good. No lord-
osis. Good colour. No faints. Not nervous.
Ht. 5 ft. 11". Wt. 11 st. 1 lb.

Urine (aft.) S.G. 1020. Albumin ($\frac{1}{2}$) and globulin
(trace). No casts, cells or oxalates. Early morning
specimen free. No oedema. No cardiac enlargement.
Arteries soft.

B.P. $\frac{130}{80}$ Erect.

$\frac{125}{80}$ Recumbent.

1923. S.R. 0. U. 2. 1 pos. (even'g).
1924. S.R. 8 days Tonsillitis. During febrile period
passed albumin and globulin (trace). 2 days
later, (afebrile) neg. U. 6. 2 pos. (even'g).
1925. S.R. 0. U. 5 all neg.
1926. S.R. 3 days feverish cold during febrile period,
passed globulin and albumin (trace). 2 days
later, (afebrile) neg. U. 5. 2 pos. (m.).
B.P. unaltered. No cardiac enlargement.
1927. S.R. 0. U. 6 1 pos. (aft.)
B.P. $\frac{130}{80}$. Arteries soft. No casts. No cardiac
enlargement. Feels well.

CASE 31. aged 23. Postman. Previous health good. No infectious disease history. Development and posture, good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 6". Wt. 10 st. 2 lbs.

Urine (m.) S.G. 1008. Albumin (haze). No globulin. No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{140}{80}$.

Recumbent $\frac{135}{80}$.

1923. S.R. 0. U. 2 both pos. (aft.).
 1924. S.R. 10 days Influenza. U. 5 all neg.
 1925. S.R. 0. U. 6 all neg.
 1926. S.R. 5 days Coryza. U. 5 - all neg.
 1927. S.R. 0. U. 6 all neg.

CASE 32. aged 19. Engine fitter. Complaint - sprain ankle. Muscular, well developed youth. Posture good. No lordosis. Colour good. Rugby football player. Not nervous. Previous health - very good. No infectious disease history.

Urine. - evening. S.G. 1018. Protein - albumin - about $\frac{1}{4}\%$ Esbach. No globulin. Casts 0. Cells 0. Oxalates 0.

Early morning specimen free.

B.P. Erect $\frac{125}{85}$

Recumbent $\frac{130}{85}$.

No oedema. No cardiac enlargement. Arteries soft.

1923. S.R. 0. U. 2. both pos. (m. 1. aft. 1).
 Specimen positive in morning (after night work) was negative, 8 hours later, after recumbency.
 1924. S.R. 6 days feverish coryza during which passed albumin (haze) and globulin (in excess). 2nd day after fall of temperature - no protein.
 1925. S.R. 10 days Influenza. U. 6 tests. 1 before rugby football match (neg), one immediately after, trace albumin, one 2 hours later neg.
 U. 3. 1 pos. (m.).
 1926. S.R. 0. U. 5 all neg.
 1927. S.R. 3 days Coryza. 8 days Influenza.
 U. 6 all neg.

CASE 33./

CASE 33. aged 20. Student. Complaint - shingles. Previous health - good. No infectious disease history. Development and posture, good. No lordosis. Good colour. Not nervous. Faints 0. Ht. 5 ft. 11 $\frac{1}{2}$ ". Wt. 12 st.

Urine (even'g). S.G. 1004. Albumin only (trace). No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$. Recumbent $\frac{125}{80}$.

1923. S.R. 0. U. 2 both neg.
 1924. S.R. 7 days Coryza. U. 7. 2 pos. (even'g).
 1925. S.R. 14 days Influenza. U. 6 - all neg.
 1926. S.R. 0. U. 6 all neg.
 1927. S.R. 5 days Dyspepsia. U. 6 - all neg.

CASE 34. aged 18. Motor-mechanic. Previous health good. No infectious disease history. Good development and posture. No lordosis. Not nervous. Good colour. Never fainted. Ht. 5 ft. 8". Wt. 11 st.

Urine (aft.) S.G. 1010. Albumin only (haze). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{75}$. Recumbent $\frac{120}{75}$.

1923. S.R. 3 days, Coryza. 5 days Enteritis. U. 2. 1 pos. (m.).
 1924. S.R. 7 days Tonsillitis. U. 6 all neg.
 1925. S.R. 5 days Coryza, 10 days Influenza. U. 5 all neg.
 1926. S.R. 3 days Coryza. U. 5 all neg.
 1927. S.R. 21 days Bronchitis. U. 6 all neg.

CASE 35. aged 25. Labourer. Complaint - Pyorrhoea. Previous health good, beyond recurrent "colds". Development - very good. No lordosis. Colour good. No infectious disease history. Not nervous. No faints. Ht. 5 ft. 8". Wt. 10 st. 13 lbs.

Urine/

Urine (morning). S.G. 1008. Albumin (haze). No globulin. No casts, or cells. A few oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{85}$ Erect. Recumbent $\frac{125}{85}$.

1923. S.R. 5 days Tonsillitis. U. 2. both neg.
 1924. S.R. 10 days Influenza. U. 5. 2 pos. (1 aft. 1 even'g).
 1925. S.R. 16 days Influenza. U. 6 all neg.
 1926. S.R. 0. U. 5. 1 pos. (m.). Specimen 6 hrs. later after recumbency, negative.
 1927. S.R. 4 days Coryza. U. 6 all neg.

CASE 36. aged 18. Motor-driver. Complaint.- bruised leg. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. No faints. Not nervous. Ht. 5 ft. 8". Wt. 10 st. 12 lbs. Amateur football player.

Urine. S.G. 1008. Protein - albumin only. Casts 0. Cells 0. Oxalates 0. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$. Recumbent $\frac{125}{85}$.

1923. S.R. 10 days Acute Tonsillitis. During febrile period passed albumin and globulin (latter in excess). 2 days later (afebrile) - protein free.
 1924. S.R. 3 days Dyspepsia. 5 days Coryza. U. 5 all neg.
 1925. S.R. 21 days Influenza. U. 5. Before a rugby match urine contained a trace of albumin. Immediately after the game - still a trace; 2 hours later, a good deal. Following day - no protein.
 1926. S.R. 0. U. 4 all neg.
 1927. S.R. 4 days Coryza. 8 days Tonsillitis. U. 5 all neg.

CASE 37. aged 25. Clerk. Good development and muscularity. Posture good. No lordosis. Not nervous. Faints 0. Previous health - good. No infectious disease history. Weight 10 st. 7 lbs. Height 5 ft. 10 ins.

Urine/

Urine. (evening) S.G. 1015. Protein present - albumin only. Not sufficient to register by Esbach. Casts 0. Cells 0. Oxalate crystals 0. Early morning specimen - free. No oedema. No cardiac enlargement.

B.P. Erect $\frac{130}{90}$ Recumbent $\frac{125}{90}$.

1923. S.R. 0. U. 3. 1 positive (m.).
 1924. S.R. 0. U. 4. 2 positive (aft. 1. even'g 1).
 1925. S.R. 10 days Influenza. U. 6. (all neg.).
 1926. S.R. 10 days Influenza. U. 5. (all neg.)
 1927. S.R. 0. U. 6 all neg.

CASE 38. aged 17. Shop assistant. Examination for entrance into a Friendly Society. Tall, rapidly growing. Ht. 5 ft. 11". Wt. 9 st. 12 lbs. Colour good. Posture good. No lordosis. Not nervous. No faints. Previous health - good. No infectious disease history.

Urine (evening). S.G. 1008. Protein - albumin only (haze). Casts - a few hyaline. Cells 0. Oxalates 0. Early morning specimen - free. Casts absent on a later examination.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{118}{75}$ Recumbent $\frac{110}{75}$.

1923. S.R. 3 days Coryza. U. 3. 1 pos. (even'g).
 1924. S.R. 10 days Influenza. U. 5 all neg.
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 10 days Coryza. U. 5. all neg.
 1927. S.R. 6 days Tonsillitis. U. 6. all neg.

CASE 39. aged 18. Printer. Complaint - "continual colds". Previous health - good until 2 years ago. No infectious disease history. Frequent attacks of Tonsillitis. Colour pastey.

Urine (aft.). S.G. 1020. Protein - albumin (2% Esbach). No globulin. Many casts - epithelial and fatty. A few red cells. No oxalates. Early morning specimen - trace albumin. Face slightly puffy. No oedema elsewhere. Arteries soft.

No cardiac enlargement. No changes in fundus.

B.P. /

B.P. $\frac{125}{90}$. Urea-content (after urea test) 1.5%.

Specific gravity somewhat fixed - varies between 1020-25. Effect of recumbency - protein sometimes goes, oftener remains. Nocturnal-diurnal rates (over several days) $\frac{D}{N} \frac{3}{2}$.

This case was considered organic.

CASE 40. aged 19. Carpenter. Previous health - good. No infectious disease. Well-developed. Posture good. No lordosis. Colour good. Not nervous. No faints. Ht. 5 ft. 7". Wt. 11 st. 2 lbs.

Urine (aft.). S.G. 1012. Albumin only (haze). Casts 0. Cells 0. Oxalates - many. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{80}$ Erect. Recumbent $\frac{125}{80}$.

- 1923. S.R. 3 days Coryza. U. 2. 1 pos. early morning after night work. 6 hours later after recumbency neg.
- 1924. S.R. 5 days Coryza. 10 days Rheumatism (non-febrile). U. 5. 2 pos. (aft.)
- 1925. S.R. 10 days Influenza. U. 4. all neg.
- 1926. S.R. 5 days Dyspepsia. U. 5. all neg.
- 1927. S.R. 0. U. 5 all neg.

CASE 41. aged 23. Motor-fitter. Complaint - dyspeptic symptoms. Previous health very good. No infectious disease history. Colour good. Strong, muscular athlete. No lordosis. Not nervous. No faints. Ht. 6 ft. 1". Wt. 12 st. 2 lbs. Plays rugby football.

Urine (morning). S.G. 1005. Albumin ($\frac{1}{4}$ %) and globulin (trace). Casts 0. Cells 0. Oxalates 0. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{135}{80}$ Erect. Recumbent $\frac{130}{80}$.

1923. /

1923. S.R. 10 days - dental extractions. U. 3. -
1 pos. in morning after night work (a. only).
After 6 hours recumbency - negative.
1924. S.R. 0. U. 6. Before a rugby football match -
a trace of globulin. Immediately after match -
neg. 2 hours later - a haze of globulin.
Following day - neg. Remainder neg.
1925. S.R. 17 days Influenza. 3 days, Cold. 5 days
Enteritis. U. 5 - all neg.
1926. S.R. 10 days - feverish coryza - during which
passed no protein. Remaining tests (6) neg.
1927. S.R. 14 days Influenza. 3 days Coryza.
U. 5 - all neg.

CASE 42. aged 18. Motor-mechanic. Previous health
good. No infectious disease history.
Development good. Posture good. No lord-
osis. Good colour. Not nervous. No
faints. Ht. 5 ft. 8". Wt. 10 st. 3 lbs.

Urine (aft.) S.G. 1016. Albumin ($\frac{1}{4}\%$) and globulin
(trace).

No casts, cells or oxalates. Early morning specimen,
free. No oedema. No cardiac enlargement. Arteries
soft.

B.P. $\frac{120}{75}$ Erect. Recumbent $\frac{115}{75}$.

1923. S.R. 7 days Tonsillitis. During febrile
period passed albumin and globulin (trace).
2 days later (afebrile) neg.
1924. S.R. 3 days Coryza. 4 days Dyspepsia. U. 6 -
1 pos. (aft.) a. only.
1925. S.R. U. 5 all neg.
1926. S.R. 0. U. 6 all neg.
1927. S.R. 10 days Boils. U. 5 - all neg.

CASE 43. aged 16. Apprentice. Came for examination
for admission to Friendly Society. Previous
health good. No infectious disease history.
Colour good. Build - no lordosis.
No faints. Athletic. Ht. 5 ft. 7".
Wt. 9 st. 2 lbs.

Urine - (evening). S.G. 1014. Protein - albumin
only (haze). Casts 0. Cells 0. Oxalates 0.

Early morning specimen - free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{115}{85}$ Erect. Recumbent $\frac{110}{85}$.

1923./

1923. S.R. 0. U. 2. 1 pos. (m).
 1924. S.R. - 3 days. Gastro-Enteritis. U. 3. all neg.
 1925. S.R. 10 days Influenza. U. 5. all neg.
 1926. S.R. 0. U. 5. all neg.
 1927. S.R. 12 days Influenza. U. 6 all neg.

CASE 44. aged 16. Apprentice. Previous health good. No infectious disease history. Development fair. Posture poor. Slight lordosis. Good colour. Faints 0. Somewhat nervous. Ht. 5 ft. 9". Wt. 10 st. 1 lb.

Urine (morning). S.G. 1004. Albumin (trace). No globulin. No casts or cells. A few oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{75}$ Recumbent $\frac{120}{75}$.

1923. S.R. 10 days Bronchitis. 4 days, Influenza. U. 2 - both neg.
 1924. S.R. 0. U. 6. 3 pos. (2 aft. 1 even'g).
 1925. S.R. 5 days Tonsillitis. During febrile period, passed to protein. U. 6 - all neg.
 1926. S.R. 10 days Influenza. U. 7 - all neg.
 1927. S.R. 21 days Catarrhal Jaundice. U. 6 - all neg.

CASE 45. aged 25. Mechanic. Complaint - cough. (pharyngeal). Development - very good. Athlete - amateur rugby football team. Previous health - good. Diphtheria, aged 12. Posture good. No lordosis. Not excitable. Good colour. No faints. Ht. 6 ft. Wt. 12 st. 2 lbs.

Urine - (morning). S.G. 1010. Albumin (small amt.) only. No casts, cells, oxalates. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$ Recumbent $\frac{125}{80}$.

1923. S.R. 5 days Gastro-Enteritis. 3 days feverish cold during which passed no protein. U. 3 other tests. 1 pos. in morning (after night work). Specimen passed 6 hours later (after recumbency) still contained a trace. Following day (m) neg.
 1924. S.R. 0. U. specimen passed before a rugby match, contained no protein. 2 specimens, one immediately after, other 2 hours after contained no protein.

1925. S.R. Influenza 10 days. U. 5 all neg.
 1926. S.R. O. U. 6 all neg.
 1927. S.R. O. U. 5 all neg.

CASE 46. aged 18. Motor fitter. Complaint - hernia. Previous health good. No infectious disease history. Development and muscularity good. Posture good. No lordosis. Good colour. Not nervous. Faints 0. Ht. 5 ft. 10". Wt. 10 st. 5 lbs.

Urine (morning). S.G. 1020. Albumin ($\frac{1}{2}\%$) and a trace of globulin. No casts, cells or oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{75}$ Erect.

Recumbent $\frac{120}{75}$.

1923. S.R. Feverish cold 5 days during which passed no protein. U. 2. both neg.
 1924. S.R. 10 days Bronchitis. U. 6. 3 pos. (2 m. 1 even'g).
 1925. S.R. O. U. 5. 1 pos. (even'g).
 1926. S.R. O. U. 6. all neg.
 1927. S.R. 3 days Coryza. 5 days Rheumatism (non-febrile). U. 3. 1 pos. (aft.). B.P. $\frac{125}{80}$.

No cardiac enlargement. Arteries soft. Feels very well. No casts.

CASE 47. aged 18. Motor-fitter. Complaint - cut finger. Previous health good. No infectious disease history. Development and posture good. No lordosis. Colour good. Not nervous. Faints 0. Ht. 5 ft. 9". Wt. 10 st. 5 lbs.

Urine (aft.) S.G. 1018. Albumin, small amt. and a trace of globulin. No casts or cells. A few oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$.

Recumbent $\frac{125}{80}$.

1923. S.R. 5 days Dyspepsia. U. 2 - both neg.
 1924. S.R. O. U. 5. 1 pos. in morning, after night work. Others neg.
 1925. S.R. 21 days Influenza. 7 days Tonsillitis, during febrile period of which, passed albumin and/

- and globulin, (latter in excess). 2 days after cessation of fever, no protein. U. 5 all neg.
1926. S.R. 0. U. 5 all neg.
1927. S.R. 3 days Coryza. U. 6 all neg.

CASE 48. aged 20. Motor mechanic. Complaint - Dyspepsia. Previous health very good. Strong, athletic. Good posture. Good colour. No lordosis. Good complexion. Not nervous. Faints 0. No infectious disease history. Ht. 5 ft. 10 $\frac{1}{2}$ ". Wt. 12 st. 1 lb.

Urine (even'g). Albumin (small amount). No globulin. No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{90}$

Recumbent $\frac{125}{90}$.

1923. S.R. 3 days feverish cold. 5 days mild Influenza. U. 2. 1 pos. aft.)
1924. S.R. 0. U. 6. all neg.
1925. S.R. 10 days Influenza. 4 days Neuralgia. U. 6. 1 pos. in morning after night work; after 6 hours recumbency - neg.
1926. S.R. 0. U. 7 - all neg.
1927. S.R. 10 days Dyspepsia. U. 5 all neg.

CASE 49. aged 17. Apprentice. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Colour good. Not nervous. No faints. Ht. 5 ft. 9 in. Wt. 10 st. 2 lbs.

Urine (even'g). S.G. 1016. Albumin only (trace). No cells, casts or oxalates. Early morning specimen, free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{115}{75}$ Erect.

Recumbent $\frac{110}{75}$.

1923. S.R. 0. U. 2. 1 pos. (aft).
1924. S.R. 0. U. 6. 1 pos. (even'g).
1925. S.R. 28 days Influenza. U. 5 all neg.
1926. /

1926. S.R. 0. U. 6 2 pos. (both m.) B.P. unaltered.
Arteries soft. No cardiac enlargement.

1927. S.R. 0. U. 6 2 pos. (1 aft. 1 even'g).

B.P. $\frac{125}{80}$ Arteries not thickened. No cardiac enlargement. Feels very well. No casts.

CASE 50. aged 17. Apprentice. Came for examination for entrance into a Friendly Society. Previous health good. No infectious disease history. Development and posture good. No lordosis. Colour good. Not nervous. No faints. Ht. 5 ft. 10". Wt. 9 st. 12 lbs.

Urine (even'g). S.G. 1016. Albumin (trace). No globulin. No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. No arterial thickening.

B.P. $\frac{130}{80}$ Erect. Recumbent $\frac{125}{80}$.

1923. S.R. 0. U. 2. both neg.

1924. S.R. 10 days Tonsillitis. U. 6 . all neg.

1925. S.R. 18 days Influenza. U. 6. 1 pos. (even'g).

1926. S.R. 0. U. 7. 2 pos. (1 aft. 1 even'g.).

B.P. unaltered. Arteries soft. No cardiac enlargement.

1927. S.R. 5 days Coryza. 5 days Coryza. U. 5 - all neg.

CASE 51. aged 22. Labourer. Complaint - deafness cerumen). Previous health good. Complexion "muddy". No infectious disease history. Development - poor. Slight degree of lordosis. Somewhat excitable. Fainted occasionally in school.

Urine (m.) S.G. 1012. Protein = albumin and globulin (g. slight only). Albumin, about $\frac{1}{4}\%$. Casts 0. Cells 0. Oxalates 0. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{95}$ Erect. Recumbent $\frac{125}{90}$

1923. /

1923. S.R. 0. U. 3. 1 pos. (even'g) - a. and g.
 1924. S.R. 14 days Influenza. 3 days Coryza.
 U. 3. 2 pos. (a. only) both even'g.
 1925. S.R. 0. U. 5 - all neg.
 1926. S.R. 9 days acute feverish cold (coryza).
 During pyrexial period - passed albumin and
 globulin (latter in excess). Both absent on
 2nd day after cessation of temperature.
 U. 5. 1 pos. (m) alb. only.
 1927. S.R. 0. U. 4 all neg.

CASE 52. aged 16. Mechanic. Came for examination
 for admission to Friendly Society. Previous
 health - good. No infectious disease
 history. Strong, muscular, athletic -
 plays for a football team. Colour good.
 Posture good. No lordosis. Faints 0.
 Not nervous. Not growing fast. Ht. 5 ft.
 4". Wt. 8 st. 10 lbs.

Urine (aft.) S.G. 1014. Albumin only ($\frac{1}{4}$). No casts,
 cells. A few oxalates. Early morning specimen, free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{80}$ Recumbent $\frac{110}{80}$.

1923. S.R. 0. U. 5. 1 pos. (even'g).
 1924. S.R. 3 days feverish coryza. U. 6. all neg.
 1925. S.R. 8 days Influenza. U. 5. 1 test, before
 a football match - no protein. Immediately
 after match - no protein. 2 hours after,
 negative. 1 test (even'g) positive.
 1926. S.R. 0. U. 4. 1 pos. (even'g). B.P. unaltered.
 1927. S.R. 3 days - Feverish Tonsillitis, during
 which passed albumin and globulin (trace). 2
 days later (afebrile) negative. 1 test later
 pos. (aft.).

B.P. $\frac{120}{80}$. No cardiac enlargement. Feels well.
 Arteries soft. No casts.

CASE 53. aged 23. Motor-fitter. Complaint -
 sprained wrist. Previous health - very
 good. Strong, muscular athlete (scratch
 man in track cycle racing). Development
 good. Posture good. No lordosis. No
 faints. Not nervous. No infectious
 disease history. Ht. 5 ft. 10". Wt. 10 st.
 8 lbs.

Urine/

Urine (even'g). S.G. 1020. Albumin - small amount. No globulin. No casts, cells or oxalates. Early morning specimen free.

B.P. $\frac{130}{85}$ Erect.

Recumbent $\frac{125}{85}$.

No oedema. No cardiac enlargement. Arteries soft.

1923. S.R. 0. U. 2 both neg.

1924. S.R. 0. U. 6. 1 pos. morning (after night work). After 6 hours recumbency specimen passed was negative. 4 remaining tests all neg.

1925. S.R. 7 days Influenza. 4 days Coryza. U. 3. (see p. 89 for results of track cycle races on the urinary protein).

1926. S.R. 7 days Coryza. U. 5. 1 pos. (even'g).

B.P. $\frac{130}{85}$. No cardiac enlargement.

1927. S.R. 16 days Dyspepsia. U. 6 all neg.

CASE 54. aged 15. Milk-boy. Complaint - sprained ankle. Previous health good except for Pneumonia in childhood. No infectious disease history. Pale, rapidly growing. Ht. 5 ft. 3 ins. Wt. 7 st. 8 lbs. Posture good. No lordosis. Not nervous. Has never fainted.

Urine - evening specimen. Protein present - albumin (a haze) and globulin (well-marked). Casts 0. Cells 0. Oxalates - a few. Early morning specimen - free.

No oedema. No cardiac enlargement. No tachycardia. Arteries soft.

B.P. Erect $\frac{120}{80}$

Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 5. 3 pos. (aft. 2. evening 1).

1924. S.R. 0. U. 3. 1 pos. (even'g). a. only.

This youth left the city at the end of the year and no further history is known.

CASE 55. aged 20. Student. Previous health good, except for dyspeptic symptoms. No infectious disease history. Colour good. Development and posture good. No lordosis. Not nervous. Ht. 5 ft. 10 $\frac{1}{2}$ ". Wt. 12 st.

Urine/

Urine (aft.) S.G. 1020. Albumin and Globulin (hazes).
 Casts 0. Cells 0. Oxalates, a few.
 Early morning specimen free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect. $\frac{120}{80}$ Recumbent.

1923. S.R. Dyspepsia 5 days. U. 2. 1 pos. (aft.).
 1924. S.R. Influenza. 10 days. U. 4. 1 pos. (even'g)
 1925. S.R. Influenza 17 days. U. 6. all neg.
 1926. S.R. 0. U. 5. all neg.
 1927. S.R. 10 days Acute Tonsillitis, during febrile
 period passed no protein. U. 6 all neg.

CASE 56. aged 20. Garage-hand. Previous health
 very good. No infectious disease history.
 Athlete - plays rugby for a local team.
 Development - very good. No lordosis.
 Not nervous. No faints. Colour good.
 Ht. 6 ft. 2". Wt. 12 st. 3 lbs.

Urine (evening). S.G. Albumin (trace). No globulin.
 No casts, cells or oxalates. Early morning specimen -
 free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect. Recumbent $\frac{115}{75}$.

1923. S.R. 6 days Dyspepsia. U. 2. 1 pos. even'g.
 1924. S.R. 0. U. 5. 1 test before rugby match
 contained a trace of albumin. Specimen passed
 immediately after match neg. One passed 2
 hours afterwards - trace albumin. Other tests
 that year. 2 pos. even'g. other neg.
 1925. S.R. 14 days Acute Tonsillitis - During febrile
 period, passed no protein. U. 4. all neg.
 1926. S.R. 10 days Bronchitis. U. 6. all neg.
 1927. S.R. 0. U. 6. all neg.

CASE 57. aged 20. Mechanic. Came for examination
 for life assurance. Previous health good.
 Development and muscularity good. Colour
 good. No lordosis. Not nervous. Height
 6 ft. 1". Wt. 10 st. 2 lbs. Never fainted.
 No infectious disease history.

Urine/

Urine (morning). S.G. 1010. Protein - albumin only (haze). Casts 0. Cells 0. Oxalates - a few.
Early morning specimen, free.
No oedema. No cardiac hypertrophy. Arteries soft.

B.P. Erect $\frac{120}{80}$

Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 2. both neg.
1924. S.R. 21 days catarrhal Jaundice. U. 6. 2 pos.
(1 m. 1 aft.)
1925. S.R. 0. U. 5. all neg.
1926. S.R. 5 days Coryza. During febrile period
passed no protein.
1927. S.R. 0. U. 6 - all neg.

CASE 58. aged 16. Apprentice (motor). Came for examination for admission into Friendly Society. Previous health - a good deal of Bronchitis, but last two years, better health. No infectious disease history. Posture and development good. No lordosis. Not nervous. Faints 0. Good colour. Ht. 5 ft. 6". Wt. 8 st.

Urine (even'g). S.G. 1014. Albumin (haze). No globulin. No casts, cells, or oxalates.
Early morning specimen - free.
No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{115}{75}$ Erect.

Recumbent $\frac{110}{75}$.

1923. S.R. 10 days, Rheumatism (non-febrile).
U. 2. all neg.
1924. S.R. 3 days Coryza. 7 days Tonsillitis.
U. 6. all neg.
1925. S.R. 12 days Influenza. U. 6. 1 pos. (morning)
after night work. Specimen passed after 6
hours recumbency, neg. 1 specimen before a
football match neg. One immediately after, and
one 2 hours after, both neg.
1926. S.R. 0. U. 5. all neg.
1927. S.R. 7 days Influenza. U. 7 all neg.

CASE 59. aged 16. Student. Complaint - erythema nodosum. Previous health - good. No infectious disease history. Development and posture, good. No lordosis. Good colour. Not nervous. Never faints, Ht. 5 ft. 7". Wt. 9 st. 8 lbs.

Urine/

Urine. S.G. 1010. Albumin only ($\frac{1}{2}\%$) No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect.

Recumbent $\frac{110}{80}$

1923. S.R. 0. U. 2. 1 sample passed in morning contained albumin. Another passed 6 hours later after recumbency, was negative.

1924. S.R. 4 days Coryza, 5 days Influenza. U. 6 - all neg.

1925. S.R. 8 days Influenza. U. 5 - all neg.

1926. S.R. 0. U. 6. 2 pos. (1 aft. 1 even'g).

B.P. $\frac{125}{80}$ No cardiac enlargement.

1927. S.R. 16 days Bronchitis. U. 5 - all neg.

CASE 60. aged 23. Wireless engineer. Complaint - constipation. Pale. Posture poor. No lordosis. Faints 0. Pneumonia in childhood. Health lately, good. Wt. 10 st. 4 lbs. Ht. 5 ft. 10". No infectious disease history. Not nervous.

Urine (morning) S.G. 1020. Protein present - both albumin ($\frac{1}{2}\%$ Esbach) and globulin, but albumin predominating. Casts 0. Cells 0. Oxalates present. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft. Blood pressure:-

Erect $\frac{125}{90}$

Recumbent $\frac{120}{90}$.

1923. S.R. 0. U. 2. 1 pos. (aft.)

1924. S.R. 0. U. 3. all positive (m. 2 aft. 1). The urine positive on one of the mornings was negative 6 hours later after rest in bed.

1925. S.R. 14 days Influenza. U. 3. all positive (aft. 2. m. 1).

1926. S.R. 0. U. 5. 2 positive (both even'g).

B.P. unaltered. No cardiac enlargement.

Arteries soft.

1927. S.R. 4 days Coryza. U. 4. 2 pos. (both aft.).

B.P. $\frac{125}{90}$. Feels very well. Arteries soft. No cardiac enlargement.

CASE 61. aged 20. Motor-mechanic. Previous health good. No infectious disease history. Development and posture, good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 7". Wt. 10 st. 2 lbs.

Urine (m). S.G. 1020. Albumin (trace). No globulin. No cells or casts. A few oxalates. Early morning specimen free.

No/

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{75}$.

Recumbent $\frac{120}{75}$.

1923. S.R. 0. U. 2 both neg.
 1924. S.R. 7 days Coryza. U. 6. 1 pos. (m). after night work. 6 hours later, same day, specimen neg. Others neg.
 1925. S.R. 0. U. 6. 2 pos. (even'g).
 1926. S.R. 3 days Coryza. 10 days Influenza. U. 7. all neg.
 1927. S.R. 7 days Coryza. U. 7 all neg.

CASE 62. aged 23. Motor-tester. Came for examination for entrance into a Friendly Society. Previous health - good. No infectious disease history. Posture and development rather poor. Slight lordosis. Not nervous. Never fainted. Colour pale. Ht. 5 ft. 10". Wt. 9 st. 10 lbs.

Urine. (aft). S.G. 1000. Albumin ($\frac{1}{2}\%$) and some globulin. No casts or cells. Some oxalates. Early morning specimen, free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{80}$ Erect.

Recumbent $\frac{115}{80}$.

1923. S.R. 6 days feverish cold, during which passed albumin and globulin (trace only). U. 2 both neg.
 1924. S.R. 5 days Coryza. 3 days Enteritis. U. 4 - 1 pos. morning (after night work). After 6 hours recumbency - neg. Others neg.
 1925. S.R. 14 days Influenza. U. 5. 2 pos. (1 aft. 1 even'g).
 1926. S.R. 0. U. 6 1 pos. aft. B.P. $\frac{125}{80}$.

No cardiac enlargement.

1927. S.R. 7 days Dyspepsia. U. 7 1 pos. (aft). B.P. unaltered. No cardiac enlargement. Arteries soft. No casts. Feels very well. Has grown into a robust young man.

CASE 63. /

CASE 63. aged 20. Motor_mechanic. Development - good. Rather pale. Posture good. No lordosis. Previous health, good. No infectious disease history. No faints. Not nervous. Ht. 5 ft. 10 ins. Wt. 10 st. 12 lbs.

Urine - S.G. 1015. Albumin ($\frac{1}{4}\%$). No globulin. No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{120}{75}$ Recumbent $\frac{115}{75}$.

1923. S.R. 0. U. 2 both neg.
 1924. S.R. 3 days Coryza. U. 6. 2 pos. (both even'g)
 1925. S.R. 54 days Scarlet Fever - see p. 95 for notes on progress of this disease and its influence on the urinary protein.
 1926. S.R. 0. U. 7. 1 pos. (even'g) when patient had a coryza cold. All others neg.

B.P. $\frac{125}{75}$. No cardiac enlargement.

1927. S.R. 0. U. 8 all neg.
 Patient is very fit and feels very well.
 B.P. unaltered. Arteries soft.

CASE 64. aged 17. Apprentice came for examination for entrance into Friendly Society. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 8. Wt. 10 st. 2 lbs.

Urine. (aft.) S.G. 1020. Albumin only (haze). No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{75}$ Erect. $\frac{115}{75}$ Recumbent.

1923. S.R. 0. U. 2. 1 pos. (aft.)
 1924. S.R. 7 days Acute Tonsillitis. During febrile period passed albumin and globulin (trace only). 2 days later (afebrile) - neg. U. 3. all neg.
 1925. /

1925. S.R. 0. U. 5. all neg.
 1926. S.R. 10 days Influenza. U. 6. 1 pos. (even'g)
 B.P. unaltered. No cardiac enlargement.
 1927. S.R. 0. U. 6. 1 pos. (morning) after night work.
 B.P. $\frac{125}{80}$. No cardiac enlargement. Arteries soft.
 No casts. Feels very well.

CASE 65. aged 25. Motor fitter. Previous health - good. No infectious disease history. Development good. Posture good. No lordosis. Not nervous. Good colour. No faints. Ht. 5 ft. 11". Wt. 12 st. 3 lbs.

Urine (m.). S.G. 1020. Albumin only (haze). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{135}{80}$.

Recumbent $\frac{130}{80}$

1923. S.R. 4 days - feverish coryza, during which passed globulin (moderate amt.) and trace albumin. 2 days later (afebrile) - urine neg. U. 1 pos. (m) after night work.
 1924. S.R. 10 days Influenza. U. 5 all neg.
 1925. S.R. 0. U. 5 2 pos. (aft).
 1926. S.R. 3 days Coryza. 5 days Tonsillitis. U. 6 all neg.
 1927. S.R. 8 days Coryza. U. 5. 2 pos. (1 aft. 1 even'g).
 B.P. $\frac{135}{80}$ No casts. No cardiac enlargement.
 Arteries soft. Feels very well.

CASE 66. aged 25. Clerk. Previous health, very good. No infectious disease history. Development very good. Rugby football player. Posture good. No lordosis. Colour good. No faints. Not nervous. Ht. 5 ft. 11½ in. Wt. 12 st. 8 lbs.

Urine (morning) S.G. 1015. Albumin - small amount. No globulin. No casts, cells, or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$

Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 4. 1 pos.(m). 3 exercise tests.
Urine. Before match, neg. Immediately after,
neg. 2 hours after, neg.
1924. S.R. 4 days Dyspepsia. 3 days Coryza. U. 6 -
all neg.
1925. S.R. 0. U.6 all neg.
1926. S.R. 0. U.7 all neg.
1927. S.R. 10 days Coryza. U.6 all neg.

CASE 67. aged 22. Chauffeur. Complaint - Colles fracture. Previous health good. No infectious disease history. Posture and development, good. No lordosis. Good colour. Not nervous. Never fainted. Ht. 5 ft. 10". Wt. 11 st. 2 lbs.

Urine (m). S.G. 1006. Albumin (trace). No globulin. No casts, cells or oxalates. Early morning specimen - free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$.

Recumbent $\frac{125}{80}$.

1923. S.R. 14 days Influenza. U. 2. 1 pos. (even'g).
1924. S.R. 0. U. 5. all neg.
1925. S.R. 4 days Dyspepsia. U. 7. 2 pos. (1 aft, 1 even'g).
1926. S.R. 0. U. 6. 2 pos. (m). B.P. unaltered. No cardiac enlargement.
1927. S.R. 5 days Coryza. U. 7. 1 pos. (aft).
B.P. $\frac{130}{80}$.

No change in position of apex beat. Arteries soft. No casts. Feels very well.

CASE 68. aged 15. Telegraph boy. Previous health good. No infectious disease history. Slightly pale. Posture poor and some degree of lordosis. Somewhat nervous. Fainted a few times at school. Ht. 5 ft. 2". Wt. 7 st. 7 lbs.

Urine (even'g). S.G. 1020. Protein - albumin and globulin (Traces only). Casts 0. Cells 0. Oxalates - a few.

Early morning specimen - free.

No oedema. No cardiac hypertrophy. Arteries soft.

B.P. /

B.P. Erect $\frac{120}{75}$ Recumbent $\frac{125}{75}$.

1923. S.R. 14 days Catarrhal Jaundice. U. 3 1 pos. (evening).
 1924. S.R. 7 days febrile Coryza, during which protein passed (albumin and globulin, latter in excess).
 2nd day after cessation of fever, protein still present (traces).
 4th day after cessation of fever, protein absent.
 U. 4. 1 pos. (m).
 1925. S.R. 14 days Influenza. U. 4. all neg.
 1926. S.R. 0. U. 4. all neg.
 1927. S.R. 0. U. 4. all neg.

CASE 69. aged 16. Apprentice. Came for examination for admission to Friendly Society. Previous health - good. No infectious disease history. Development good. Good posture. No lordosis. Not nervous. Never faints. Colour good. Ht. 5 ft. 7". Wt. 9 st. 2 lbs.

Urine. (even'g). S.G. 1012. Protein - albumin only (small amount). Cells 0. Casts 0. Oxalates 0. Early morning specimen free. No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{115}{80}$ Recumbent $\frac{110}{80}$.

1923. S.R. 5 days - coryza. U. 2. 1 post. (aft.)
 1924. S.R. 10 days Acute Tonsillitis, during which passed albumin chiefly (trace globulin).
 2 days later (afebrile) - trace albumin.
 2 days later neg.
 1925. S.R. 21 days Influenza. 3 days Coryza.
 U. 5. all neg.
 1926. S.R. 0. U. 6 all neg.
 1927. S.R. 10 days Tonsillitis. 5 days Dyspepsia.
 U. 7. all neg.

CASE 70. aged 17. Student. Complaint - sprained ankle. Development - moderate. Posture good. No lordosis. Good colour. Not nervous. Ht. 5 ft. 11 $\frac{1}{2}$ ". Wt. 12 st. 2 lbs. Never fainted. Previous health good. No infectious disease history.

Urine. /

Urine. (morning). S.G. 1020. Protein - albumin only (haze). Casts 0. Cells 0. Oxalates 0.
No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect.

Recumbent $\frac{120}{80}$.

1923. S.R. 10 days Bronchitis. U. 2. 1 pos. (even'g)
1924. S.R. 5 days Coryza. 7 days Influenza.
U. 6. all neg.
1925. S.R. 10 days Multiple Boils. U. 5. all neg.
1926. S.R. 0. U. 6. all neg.
1927. S.R. 6 days Feverish Coryza. U. 5. all neg.

CASE 71. aged 16. Schoolboy. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Good colour. Not nervous. Faints 0.
Ht. 5 ft. 3". Wt. 7 st. 12 lbs.

Urine. (m) S.G. 1016. Albumin ($\frac{1}{4}$) and globulin (trace). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{80}$

Recumbent $\frac{115}{80}$.

1923. S.R. 10 days Influenza. U. 2. 1 pos. (even'g). alb. only.
1924. S.R. 12 days Quinsy. U. 5. 1 pos. (even'g).
1925. S.R. 0. U. 4. all neg.
1926. S.R. 0. U. 5. 1 pos. (even'g).
B.P. unaltered. No cardiac enlargement.
1927. S.R. 0. U. 6. 2 pos. (aft. 1. even'g. 1).

B.P. $\frac{120}{80}$. No cardiac enlargement. Arteries soft.

No casts. Feels very well. Is now a strong, well developed young man.

CASE 72. aged 16. Foundry work. Complaint - alopecia areata. Previous health - "rather delicate". Pale. Poor posture and some lordosis. Somewhat nervous. No faints. No infectious disease history. Ht. 5 ft. 4". Wt. 7 st. 8 lbs.

Urine/

Urine (evening) S.G. 1014. Protein - albumin (about $\frac{1}{4}\%$) and globulin (haze only). Casts 0. Cells 0. Oxalates - a few. Early morning specimen - free. No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{110}{80}$.

Recumbent $\frac{105}{80}$.

1923. S.R. 0. U. 3. 1 positive (even'g) g. only.
 1924. S.R. 4 days feverish cold. Urine during period of pyrexia contained albumin and globulin (latter in excess). 2 days later (afebrile) - free.
 1925. S.R. 0. U. 4. all neg.
 1926. S.R. 0. U. 5. all neg.
 1927. S.R. 10 days, Dyspepsia. U. 6. all neg.

CASE 73. aged 18. Clerk. Development good. Rather overgrown. Ht. 6 ft. 2". Wt. 12 st. Posture - poor. Slight lordosis. Not nervous. Never fainted. Previous health good. No infectious disease history.

Urine (aft.) Protein present in small amount. Albumin only. Casts 0. Cells 0. Oxalates 0. S.G. 1015. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{118}{80}$

Recumbent $\frac{110}{80}$.

1923. S.R. 10 days Bronchitis. U. 2. 1 pos. (even'g).
 1924. S.R. 12 days Acute Coryza. During febrile period passed both albumin and globulin (latter in excess). 2 days after drop of fever - no protein found.
 1925. Dyspepsia - 6 days. U. 5. all neg.
 1926. S.R. 10 days Influenza. U. 5. all neg.
 1927. S.R. 3 days Coryza. 10 days Furunculosis. U. 4. all neg.

CASE 74. aged 18. Motor-driver. Previous health - very good. No infectious disease history. Muscularity and development good. No lordosis. Not nervous. Never fainted. Ht. 5 ft. 10". Wt. 10 st. 12 lbs. Colour good.

Urine/

Urine. (m). S.G. 1010. Protein, haze albumin only.
 Casts 0. Cells 0. Oxalates - many.
 Early morning specimen - free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{90}$ Erect. Recumbent $\frac{125}{85}$.

1923. S.R. 6 days Coryza. 10 days Acute Tonsillitis.
 U. 2. 1 pos. (m).
 1924. S.R. 0. U. 5. 1 pos. (m) albumin, - a good deal.
 1925. S.R. 10 days Gastro-Enteritis. U. 6 - all neg.
 1926. S.R. 0. U. 5. all neg.
 1927. S.R. 10 days Acute Tonsillitis - during pyrexial
 period, no protein passed.

CASE 75. aged 19. Engine-fitter. Came for examination for entrance into Friendly Society. Previous health - good. No infectious disease history. Development and muscularity good. Rugby player. Posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 6 ft. 2". Wt. 12 st. 2 lbs.

Urine (even'g). S.G. 1016. Albumin ($\frac{1}{4}\%$). No globulin. No cells, casts, or oxalates. Early morning specimen free.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$ Recumbent $\frac{120}{80}$.

1923. S.R. 4 days Coryza. U. 3. 1 pos. m. (after night work). After 6 hours recumbency - still a trace albumin. Following day (m). neg.
 1924. S.R. 0. U. 7. all neg.
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 0. U. 6. one positive (trace albumin). before a rugby match. Specimen immediately after match, neg. Another 2 hours later, also neg.

B.P. $\frac{125}{80}$. No cardiac enlargement.

- Feels very well. Plays rugby regularly.
 1927. S.R. U. 7. all neg.

CASE 76. aged 15. School-boy. Complaint, epistaxis. Pale. Overgrown. Poor posture, some lordosis. Somewhat excitable. Previous health - never very robust, but not definite ailments. No infectious disease history. Never fainted. Ht. 5 ft. 10". Wt. 10 st.

Urine (even'g). Albumin only. S.G. 1008. Casts 0.
Cells 0. Oxalates - a few.
Early morning specimen free.
No oedema. No cardiac enlargement. No arterial thickening.

B.P. Erect $\frac{115}{80}$

Recumbent $\frac{110}{80}$.

1923. S.R. 3 days feverish coryza, during which passed albumin and globulin (latter in excess). 2 days later (afebrile) neg.
1924. S.R. 14 days Bronchitis. U. 6. 0.
1925. S.R. 21 days Influenza. U. 7. 1 pos. (in morning, after night work. After 6 hours recumbency - neg).
1926. S.R. 0. U. 6. 1 pos. (even'g). B.P. unaltered.
1927. S.R. 5 days coryza. U. 7. 2 pos. (1 aft. 1 evening).

B.P. $\frac{120}{80}$. Feels very well. is a broad muscular youth now. Arteries soft. No cardiac enlargement. No casts.

CASE 77. aged 24. Engine-mechanic. Complaint - sprained ankle. Previous health - very good. No infectious disease history. Muscular development - rather poor. Colour pale. No lordosis. No faints. Weight 10 st. 1 lb. Ht. 5 ft. 7".

Urine. S.G. 1006. Albumin only (trace). No casts, or cells. A few oxalates.
Early morning specimen free.
No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{80}$ Erect.

Recumbent $\frac{120}{85}$.

1923. S.R. 0. U. 2. 1 pos. (even'g).
1924. S.R. 10 days Influenza. 3 days feverish coryza, during which passed albumin and globulin (trace only). 2 days later - free. U. 4. 2 post. (1 aft. 1 even'g).
1925. S.R. 0. U. 5. 1 pos. (even'g).
1926. S.R. 10 days Bronchitis. 3 days Coryza. U. 6. 2 pos. (1 aft. 1 even'g).
B.P. unaltered. No cardiac enlargement.
1927. S.R. 0. U. 5. all neg.

CASE 78. /

CASE 78. aged 15. School-boy. Complaint - Impetigo. Previous health, good. No infectious disease history. Development average. Posture good. No lordosis. Colour good. Not nervous. Never faints. Ht. 5 ft. 4". Wt. 7 st. 8 lbs.

Urine (morning) S.G. 1010. Albumin (trace). No globulin. No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{110}{75}$

Recumbent $\frac{110}{75}$.

1923. S.R. 0. U. 2. both neg.
 1924. S.R. 8 days Tonsillitis. U. 6 all neg.
 1925. S.R. 0. U. 7. 1 pos. (m).
 1926. S.R. 13 days Bronchitis. U. 6. all neg.
 1927. S.R. 20 days Influenza. U. 7. all neg.

CASE 79. aged 19. Motor-mechanic. Previous health good. No infectious disease history. Development and posture, good. Not nervous. No lordosis. Good colour. Faints - 0. Ht. 5 ft. 7". Wt. 10 st. 6 lbs.

Urine (m). S.G. 1018. Albumin only (trace). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{75}$

Recumbent $\frac{115}{75}$.

1923. S.R. 7 days Tonsillitis. During febrile period passed globulin and trace albumin. 2 days later - no protein. U. 2. both pos. (m).
 1924. S.R. 3 days Coryza. 4 days Dyspepsia. U. 5 - all neg.
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 5 days Coryza. U. 6. all neg.
 1927. S.R. 0. U. 6 all neg.

CASE 80. aged 17. Complaint. Sprained ankle. Previous health - good. Ht. 5 ft. 3". Wt. 7 st. 12 lbs. Good colour. Posture good. No lordosis. No faints. Not nervous. No history of any infectious diseases.

Urine. /

Urine. S.G. 1000. Protein - albumin only, haze.
 Casts 0. Oxalates 0. Cells 0.
 No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{115}{85}$ Erect. Recumbent $\frac{110}{85}$.

1923. S.R. 3 days Coryza. 3 days Coryza. U. 4 - all neg.
 1924. S.R. 5 days Tonsillitis. During febrile period passed albumin and globulin (a. predominating). 2 days after cessation of fever - no protein.
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 5 days Dyspepsia. U. 4. all neg.
 1927. S.R. 10 days Influenza. U. 6. all neg.

CASE 81. aged 16. Messenger. Complaint - Impetigo. Previous health good. No infectious disease history. Somewhat pale. Growing fast. Posture rather poor, slight lordosis. Somewhat nervous. Fainted occasionally when younger. Ht. 5 ft. 10". Wt. 8 st. 2 lbs.

Urine. S.G. 1015. Protein - albumin ($\frac{1}{2}\%$ Esbach) and a trace of globulin. Casts 0. Cells 0. Oxalates 0. Early morning specimen - free.
 No oedema or cardiac enlargement. Arteries soft.

B.P. Erect $\frac{110}{75}$ Recumbent $\frac{110}{75}$.

1923. S.R. Multiple boils, 14 days. Coryza, 6 days. U. 2. 1 pos. (even'g).
 1924. S.R. 0. U. 5. 1 pos. (m).
 1925. S.R. 10 days Acute Tonsillitis. During febrile period passed albumin and a small haze of globulin. 2 days after cessation of temperature a little albumin still present. 2 days later no protein found.
 1926. S.R. 0. U. 5. all neg.
 1927. S.R. 10 days Influenza. U. 6. 2 pos. (1 aft. 1 even'g). a. only. No casts.

B.P. $\frac{120}{80}$. No cardiac hypertrophy. Arteries soft.

Feels very well.

CASE 82. aged 25. Grinder. Complaint - Diarrhoea. previous health good. No infectious disease history. Development and posture good. No lordosis. Good colour. Not nervous. /

nervous. Faints 0. Ht. 5 ft. 11". Wt. 12 st. 3 lbs.

Urine. (morning). S.G. 1020. Albumin only (haze). No casts, cells, or oxalates. Early morning specimen, free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$

Recumbent $\frac{125}{80}$

1923. S.R. 0. U. 2. 1 pos. (m) after night work
1 pos. (aft.).

1924. S.R. 0. U. 5. 1 pos. (m) after night work.
Others neg.

1925. S.R. 0. U. 6. all neg.

1926. S.R. 10 days Tonsillitis. During febrile period, passed albumin and globulin (latter in excess). 2 days after fall of temp. urine, neg. B.P. $\frac{130}{80}$.

1927. S.R. 10 days Coryza. U. 6. all neg.

CASE 83. aged 18. Furnace-man. Complaint - strained shoulder. Previous health, good. No infectious disease history. Development and posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 6". Wt. 10 st. 7.

Urine (aft). S.G. 1020. Albumin only (haze). No casts, or cells. Many oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{80}$

Recumbent $\frac{115}{80}$.

1923. S.R. 10 days Influenza. 3 days Diarrhoea.
U. 2. 1 pos. (aft).

1924. S.R. 10 days Tonsillitis, during febrile period passed albumin and globulin (trace only) 2 days later, absent. U. 6 3 pos. (2 aft. 1 m.)

1925. S.R. U. 6. 1 pos. morn. (after night work).

1926. S.R. 3 days Coryza. 7 days Bronchitis.
U. 6 - all neg.

1927. S.R. 0. U. 6 all neg.

CASE 84. /

CASE 84. aged 25. Store-keeper. Complaint - Psoriasis. Previous health good. No other infectious disease. Well developed. Posture good. No lordosis. Ht. 5 ft. 11". Weight 12 st. 2 lbs. No faints. Not nervous.

Urine - morning specimen. Albumin (about $\frac{1}{4}\%$). No globulin. Casts 0. Cells 0. S.G. 1010. Oxalates present. Early morning specimen - protein free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{135}{85}$ Recumbent $\frac{130}{80}$.

1923. S.R. 0. U. 2. 1 pos. (even'g).
 1924. S.R. 0. U. 3. 2 pos. (1 even'g. 1 aft.).
 1925. S.R. 0. U. 2. 2 pos. (both even'g).
 1926. S.R. 7 days Influenza. U. 3. 1 pos. (m).
 No change in clinical condition.
 1927. S.R. 0. U. 3. 1 pos. (even'g).

B.P. $\frac{135}{85}$. Feels very well. No cardiac enlargement. Arteries soft. No casts.

CASE 85. aged 24. Motor-fitter. Complaint - dyspepsia. Development, very good. Posture good, no lordosis. Previous health - appendicitis 1918 - otherwise good. No infectious disease history. Colour good. Not nervous. Ht. 5 ft. 10". Wt. 12 st. 1 lb.

Urine (morning). S.G. 1006. Albumin - small amt. No globulin. No casts or cells. A few oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{125}{80}$ Erect. Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 2 both neg.
 1924. S.R. 6 days Tonsillitis. 3 days Diarrhoea. U. 5. 1 pos. morning (after night work), Evening of same day, after rest in bed, neg. Others neg.
 1925. S.R. 5 days Influenza. U. 6. all neg.
 1926. S.R. 0. U. 6 all neg.
 1927. S.R. 3 days Coryza. 5 days sciatic pain. U. 7 all neg.



CASE 86. aged 25. Motor Mechanic. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Not nervous. No faints. Good colour. Ht. 6 ft. Wt. 12 st. 2 lbs.

Urine (aft.) S.G. 1004. Albumin only ($\frac{1}{4}$). No casts, cells or oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$

Recumbent $\frac{125}{80}$.

1923. S.R. 0. U. 2. both pos. (m).
 1924. S.R. 8 days Bronchitis (mild). 3 days Coryza.
 U. 4 all neg.
 1925. S.R. 0. U. 6 all neg.
 1926. S.R. 5 days, Boils. U. 5. all neg.
 1927. S.R. 0. U. 6. 2 pos. (even'g).

B.P. $\frac{130}{80}$. No cardiac enlargement. Arteries soft. No casts.

Feels very well.

CASE 87. aged 17. Shop-assistant. Previous health good. No infectious disease history. Development good. Posture good. No lordosis. Good colour. Faints 0. Somewhat nervous. Ht. 5 ft. 8". Weight 10 st. 10 lbs.

Urine (even'g). S.G. 1018. Albumin (trace). No globulin. No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{118}{80}$

Recumbent $\frac{110}{80}$.

1923. S.R. 0. U. 2. 1 pos. (aft.)
 1924. S.R. 7 days Coryza. U. 7. all neg.
 1925. S.R. 21 days Influenza and Bronchitis.
 U. 6. all neg.
 1926. S.R. 0. U. 6. all neg.
 1927. S.R. 10 days Tonsillitis. U. 7. all neg.

CASE 88. /

CASE 88. aged 19. Baker. Complaint - deafness (cerumen). Previous health - good. No infectious disease history. Development and posture good. Colour pale. No lordosis. Not nervous. Faints 0. Ht. 5 ft. 8". Wt. 9 st. 12 lbs.

Urine - (m). S.G. 1020. Albumin only (Trace). No casts, cells or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{75}$ Recumbent $\frac{120}{75}$.

1923. S.R. 12 days Orchitis. U. 2. 1 pos (aft.)
 1924. S.R. 0. U. 6. all neg.
 1925. S.R. 21 days Influenza. U. 5. 2 pos. (1 m. 1 even'g).
 1926. S.R. 0. U. 6. all neg.
 1927. S.R. 10 days Bronchitis 2 days Coryza. U. 6. all neg.

CASE 89. aged 19. Lathe-work. Complaint - cut hand. Previous health good. No infectious disease. Development and posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 8 $\frac{1}{2}$ ". Wt. 10 st. 3 lbs.

Urine (morning). S.G. 1008. Albumin (small amount). No globulin. No casts, cells or oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{80}$ Recumbent $\frac{115}{80}$.

1923. S.R. 3 days coryza. U. 2. 1 pos. (morning). Specimen 6 hrs. later (with recumbency) was negative.
 1924. S.R. 10 days Bronchitis. U. 6. 3 pos. (2 even'g. 1 m.).
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 8 days Quinsy. U. 6 all neg.
 1927. S.R. 0. U. 5. all neg.

CASE 90. aged 15. Message-boy. Examined for admission into a Friendly Society. Previous health good. No infectious disease history. Colour good. Posture poor. Slight lordosis. Never/

Never faints. Not nervous. Ht. 5 ft. 3".
Wt. 7 st. 5 lbs.

Urine (m). S.G. 1005. Protein, albumin (Esbach $\frac{1}{8}\%$) and globulin (slight). Casts 0. Cells 0. Oxalates 0. No oedema. No cardiac enlargement. Heart, slow (80). Arteries soft. Early morning specimen - free.

B.P. Erect $\frac{115}{75}$.

Recumbent $\frac{115}{75}$.

1923. S.R. 10 days Acute Tonsillitis. 3 days Coryza. During febrile period passed albumin and globulin (latter predominating). 2 days after fall of temp. passed globulin only. 2 days later - no protein.
1924. S.R. 10 days - feverish Coryza. U. 5. 1 pos. (even'g).
1925. S.R. 0. U. 3. 1 pos. in early morning after night work. 6 hours later after rest in bed, neg.
1926. S.R. 21 days Influenza. U. 5. all neg.
1927. S.R. 21 days, Bronchitis. U. 6. all neg.

CASE 91. aged 24. Shop-assistant. Complaint - Dyspepsia. Previous health - Scarlet Fever and Diphtheria 7 years ago. Never been very well since. He has been absent from work a good deal in the last few years.

Urine - (morning). S.G. 1025. Albumin (.3%). No globulin. Casts - epithelial and fatty. Cells 0. No oedema. Slight cardiac enlargement. Apex beat in 6" space, outside mid-clavicular line. Arteries somewhat thickened.

Early morning sample contains a trace of albumin. D $\frac{2}{2}$. Specific gravity of various samples of urine N $\frac{2}{2}$ passed during the day varies only 7 points, i.e. some fixation of specific gravity. No urinary specimen has even been completely free from albumin.

Concentrates urea (Maclean test) 1.4%. This case is definitely organic and was not included in the study of the benign cases.

CASE 92. aged 17. Motor-mechanic. Complaint - acne. Previous health good. No infectious disease history. Development and muscularity very good. Rugby football player. Posture good. No lordosis. Good colour. Not nervous. Never fainted. Ht. 5 ft. 10". Wt. 11 st. 2 lbs.

Urine/

Urine. (even'g). S.G. 1004. Albumin only (trace).
No casts or cells. Many oxalate crystals.
Early morning specimen, free.
No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{75}$

Recumbent $\frac{110}{75}$.

1923. S.R. 0. U. 2. 1 pos. early morning (after night work). Evening of same day (after sleep) neg.
1924. S.R. 3 days Coryza. Urine tested before a rugby match - no protein. At end of match - neg. 2 hours after also neg. U. 3. 1 pos. (aft.)
1925. S.R. 7 days Tonsillitis. U. 6. all neg.
1926. S.R. 0. U. 5. all neg.
1927. S.R. 10 days Bronchitis. U. 6. all neg.

CASE 93. aged 17. Porter. Previous health, good. No infectious disease history. Development and posture good. No lordosis. Not nervous. No faints. Good colour. Ht. 5 ft. 7". Wt. 9 st. 5 lbs.

Urine (even'g). S.G. 1012. Albumin ($\frac{1}{2}\%$) and also globulin. No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{120}{80}$ Erect.

Recumbent $\frac{115}{80}$.

1923. S.R. 0. U. 2. 1 pos. (aft.) albumin only.
1924. S.R. 5 days Coryza. U. 6. 1 pos. (even'g).
1925. S.R. 12 days. Influenza. U. 7. all neg.
1926. S.R. 0. U. 3. all neg.
1927. S.R. 7 days Coryza. U. 6. all neg.

CASE 94. aged 23. Clerk. Previous health - 2 attacks of pneumonia (childhood) otherwise good. No infectious disease history. Development good. Posture good. No lordosis. Good colour. Not nervous. Faints 0. Ht. 6 ft. 1". Wt. 12 st. 12 lbs.

Urine (m) S.G. 1012. Albumin (trace) No globulin. No cases or cells. A few oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{130}{80}$

Recumbent $\frac{125}{80}$

1923. S.R. 7 days Tonsillitis; during febrile period passed no protein. U. 2. both neg.
 1924. S.R. 0. U. 6. 2 pos. (both even'g).
 1925. S.R. 8 days Influenza. U. 5. all neg.
 1926. S.R. 0. U. 6. all neg.
 1927. S.R. 10 days Dyspepsia. U. 7. all neg.

CASE 95. aged 20. Motor-mechanic. Previous health very good. Development and posture good. No lordosis. Good colour. No infectious disease history. Not nervous. No faints. Ht. 5 ft. 11". Wt. 11 st. Rugby football player.

Urine - even'g. S.G. 1008. Albumin only (Trace). No casts, cells or oxalates. Early morning specimen free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$

Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 2. 1 pos. (even'g).
 1924. S.R. 3 days Coryza. Exercise test. Before a rugby football match urine was free from protein. Immediately after end of match - albumin (small amt.). 2 hours after - trace albumin. Following day - no albumin. U. 5. 2 pos. (even'g).
 1925. Developed acute appendicitis in March and died after operation, of general peritonitis.

CASE 96. aged 15. Apprentice. Previous health, Appendicitis aged 10, otherwise very good. No infectious disease history. Good colour. Development and muscularity fair. No lordosis. Slightly nervous. No faints. Ht. 5 ft. 3". Wt. 7 st. 8 lbs.

Urine - (morning). S.G. 1014. Protein - albumin only (about $\frac{1}{4}$ Esbach). Casts 0. Cells 0. Oxalates 0. Early morning specimen - free.

B.P. Erect $\frac{125}{85}$

Recumbent $\frac{120}{85}$

No oedema. No cardiac enlargement. No arterial thickening.

1923. S.R. 4 days feverish cold. 6 days Dyspepsia. U. 2. 1 pos. (aft). Urine pos. in the morning was free in a specimen passed after 6 hours recumbency.

1924. /

1924. S.R. 10 days Bronchitis. U. 5. all neg.
 1925. S.R. 0. U. 6. all neg.
 1926. S.R. 10 days Influenza. 3 days rheumatism
 (non-febrile). U. 5. all neg.
 1927. S.R. 3 days Coryza. U. 6. all neg.

CASE 97. aged 18. Apprentice. Complaint - styes.
 Previous health good. No infectious
 disease history. Development and posture
 good. No lordosis. Good colour. Not
 nervous. No faints. Ht. 5 ft. 4".
 Wt. 9 st. 10 lbs.

Urine. (m). S.G. 1020. Albumin ($\frac{1}{2}\%$) and globulin
 (trace)

No cells, casts or oxalates. Early morning specimen
 free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{120}{75}$

Recumbent $\frac{115}{75}$.

1923. S.R. 5 days Dyspepsia. U. 2. both neg.
 1924. S.R. 0. U. 6. 2 pos. (both even'g).
 1925. S.R. 0. U. 5. all neg.
 1926. S.R. 18 days Bronchitis. U. 6. 2 pos. (both m.)

B.P. $\frac{125}{80}$. No cardiac enlargement.

1927. S.R. 0. U. 7. 1 pos. (even'g). B.P. unaltered.
 Arteries soft. No cardiac enlargement.
 Feels very well. Grown into a fine, muscular
 young man.

CASE 98. aged 15. School-boy. Previous health good.
 Development, poor. Some lordosis. No
 infectious disease history. Colour pale.
 Somewhat nervous. Faints occasionally.
 Ht. 5 ft. 5 in. Wt. 7 st. 8 lbs.

Urine. S.G. 1000. Albumin ($\frac{1}{4}\%$) and globulin (trace).
 A few oxalates. No casts or cells. Early morning
 specimen free.

No oedema. No cardiac enlargement. No arterial
 thickening.

B.P. Erect $\frac{115}{75}$

Recumbent $\frac{110}{75}$.

1923. /

1923. S.R. 3 days Coryza. U. 2. both neg.
 1924. S.R. 11 days Influenza. U. 6. all neg.
 1925. S.R. 28 days Influenza. U. 5. 1 pos. (even'g).
 1926. S.R. 0. U. 6. all neg.
 1927. S.R. 0. U. 5. all neg.

CASE 99. aged 15. School-boy. Previous health good. No infectious disease history. Posture and development, good. No lordosis. Good colour. Never faints. Not nervous. Ht. 5 ft. 2". Wt. 7 st.

Urine (aft). S.G. 1010. Albumin ($\frac{1}{4}\%$). No globulin. No casts, cells or oxalates. Early morning specimen, free.

No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{75}$ Recumbent $\frac{110}{75}$.

1923. 3 days Coryza. 7 days, Influenza. U. 2. - both neg.
 1924. S.R. 10 days Tonsillitis. U. 7. 3 pos. (all aft.)
 1925. S.R. 0. U. 6. 1 pos. (aft.)
 1926. S.R. 10 days Influenza. 4 days Enteritis. U. 6. all neg.
 1927. S.R. 0. U. 5. all neg.

CASE 100. aged 24. Clerk. Complaint - sycosis barbae. Previous health - dyspepsia. Otherwise very healthy. Development and posture - average. No lordosis. No infectious disease history. Colour good. Not nervous. Ht. 5 ft. 6". Wt. 10 st. 2 lbs.

Urine (aft.) Albumin in small amount. No casts, cells or oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{130}{85}$ Erect. Recumbent $\frac{125}{80}$.

1923. S.R. 10 days, Sciatica. U. 2. both neg.
 1924. S.R. 0. U. 7. 2 pos. (even'g).
 1925. S.R. 14 days, Influenza. U. 5. all neg.
 1926. S.R. 0. U. 6. all neg.
 1927. S.R. 10 days, Dyspepsia. 4 days, Coryza. U. 5. all neg.

CASE 101. aged 17. Garage-hand. Complaint.
Health record good. Well-developed,
muscular youth. Posture good. No lordosis.
No infectious disease history. Colour
good. Not nervous. Never fainted.
Ht. 5 ft. 10". Wt. 10 st. 5 lbs.

Urine S.G. 1015. Protein - albumin only. No casts,
cells or oxalates. Early morning specimen - free.
No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{80}$ Recumbent $\frac{120}{80}$.

1923. S.R. 0. U. 2. 1 pos. (m).
1924. S.R. 3 days - feverish coryza, during which
passed albumin and globulin (a. in excess).
2 days later - no protein.
1925. S.R. 6 days Coryza. U. 5. 1 pos. (m).
6 hours later after recumbency - protein free.
1926. S.R. 6 days - feverish coryza. 10 days
Influenza; during febrile period of which urine
contained no protein. U. 4. all neg.
1927. S.R. 10 days Bronchitis. U. 6. all neg.

CASE 102. aged 16. Carpenter. Came for examination
for admission to a Friendly Society.
Previous health good. No infectious
disease history. Posture good. No lordosis.
Not growing fast. Good colour. Not nervous.
Ht. 5 ft. 3". Wt. 7 st. 10 lbs.

Urine (even'g). S.F. 1000. Protein - albumin only
(small amount). Casts 0. Cells 0. Oxalates 0.
Early morning specimen free.
No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{80}$ Recumbent $\frac{120}{80}$

1923. S.R. 6 days Gastro-Enteritis. U. 2. 1 pos.
(even'g).
1924. S.R. U. 6. 2 pos. (even'g).
1925. S.R. 21 days Influenza. U. 5. all neg.
1926. S.R. 6 days Otitis. 3 days Enteritis.
U. 6. all neg.
1927. S.R. 0. U. 6. all neg.

CASE 103.

CASE 103. aged 18. Student. Previous health - good. No infectious disease history. Rather overgrown. Pale. Posture - average. No lordosis. Not nervous. No faints. Colour good. Ht. 6 ft. 1". Wt. 11 st. 1 lb.

Urine (aft). S.G. 1012. Albumin ($\frac{1}{4}\%$) and globulin (trace). No casts, or cells. A few oxalates.

B.P. Erect $\frac{120}{80}$

Recumbent $\frac{120}{80}$.

No oedema. No cardiac enlargement. Arteries soft.

1923. S.R. 7 days acute (febrile) follicular tonsillitis during which passed no protein.

U. 2. both neg.

1924. S.R. 10 days Bronchitis. U. 6. all neg.

1925. S.R. 18 days Influenza. 10 days Tonsillitis.

U. 7. all neg.

1926. S.R. 0. U. 6. all neg.

1927. S.R. 3 days Coryza. 4 days Dyspepsia.

U. 6. all neg.

CASE 104. aged 15. School-boy. Came for examination for entrance into Friendly Society. Previous health good. No infectious disease history. Posture rather poor, with some degree of lordosis. Good colour. Slightly nervous. Never fainted. Ht. 5 ft. 3". Wt. 7 st. 10 lbs.

Urine. (aft.) S.G. 1008. Albumin only (haze). No cells, casts, or oxalates. Early morning specimen, free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{75}$

Recumbent $\frac{115}{75}$.

1923. S.R. 0. U. 2. both neg.

1924. S.R. 7 days Influenza. 3 days Tooth-ache.

U. 6. 3 pos. (all even'g).

1925. S.R. 13 days Influenza. U. 7. all neg.

1926. S.R. 0. U. 6. all neg.

1927. S.R. 10 days Influenza. U. 7. all neg.

CASE 105. /

CASE 105. aged 25. School-teacher. Previous health good. No infectious disease history. Development and muscularity good. Good posture. No lordosis. Colour good. Not nervous. No faints. Ht. 5 ft. 11". Wt. 12 st. 1 lb.

Urine (aft). S.G. 1010. Albumin only (trace). No cells, casts or oxalates. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{125}{75}$ Recumbent $\frac{120}{75}$.

1923. S.R. 3 days Coryza. U. 2. both neg.
 1924. S.R. 10 days Rheumatism. U. 5. 2 pos. (even'g)
 1925. S.R. 12 days Influenza. U. 6. 1 pos. (m).
 1926. S.R. 0. U. 7. 2 pos. (m). B.P. unaltered.
 No cardiac enlargement. Arteries soft.
 1927. S.R. 10 days Acute Tonsillitis; during febrile period albumin (traces) and globulin (in excess). 2 days later (afebrile) urine neg. U. 5. - 2 pos. (both even'g). No casts. B.P. $\frac{125}{80}$.
 No cardiac enlargement. No arterial thickening. Feels very fit.

CASE 106. Motor-fitter. Aged 18. Previous health - 2 attacks of Pneumonia. Otherwise good. Posture good. No lordosis. Good colour. Not nervous. No faints. Ht. 5 ft. 9". Wt. 10 st. 2 lbs.

Urine (m). S.G. 1015. Protein - albumin ($\frac{1}{4}$ " Esbach) and globulin. Casts 0. Cells 0. Oxalates - a few. Early morning specimen free. No oedema. No cardiac enlargement. Arteries soft.

B.P. $\frac{135}{85}$ Erect. Recumbent $\frac{130}{85}$.

1923. S.R. 10 days Muscular Rheumatism. (non-febrile). 4 days Coryza. U. 2. 1 pos. (m).
 1924. S.R. 0. U. 2. both neg.
 1925. S.R. 10 days Influenza. U. 5. 1 pos. early morning after night work. After 6 hours recumbency - no protein. Remaining 3, all neg.
 1926. S.R. 4 days Dyspepsia. 3 days Coryza. U. 5. all neg.
 1927. S.R. 0. U. 6. all neg.

CASE 107. aged 16. Message-boy. Complaint - sprained ankle. Previous health good. No infectious disease history. Development and posture, poor. Some slight lordosis. Good colour. Never faints. Slightly nervous. Growing fast. Ht. 5 ft. 9". Wt. 8 st. 7 lbs.

Urine (morning). S.G. 1020. Albumin only ($\frac{1}{4}\%$). No globulin. No casts, cells, or oxalates. Early morning specimen - free. No oedema. No cardiac enlargement. Arteries soft.

B.P. Erect $\frac{115}{80}$

Recumbent $\frac{110}{75}$.

1923. S.R. 5 days Coryza. 4 days Gastro-Enteritis. U. 2. both neg.
 1924. S.R. 8 days Influenza. U. 5. all neg.
 1925. S.R. 0. U. 6. all neg.
 1926. S.R. 10 days Acute tonsillitis during which passed albumin (trace) and globulin in excess. 2 days after cessation of temperature - glob. only. 2 days later absent. Later in year U. 2. both neg.

B.P. $\frac{120}{80}$.

No cardiac enlargement.

1927. S.R. 10 days Coryza. U. 5. all neg.

FREQUENCY AND AGE INCIDENCE.

There is no disagreement among investigators as to the frequency of the condition during early life. As to actual statistics, however, there are wide differences.

The largest investigation (numerically) was that of Maclean¹¹ on 60,000 recruits (average age 19) of the British draft in the Great War, in which albumin unassociated with other defects was found in 5% of cases.

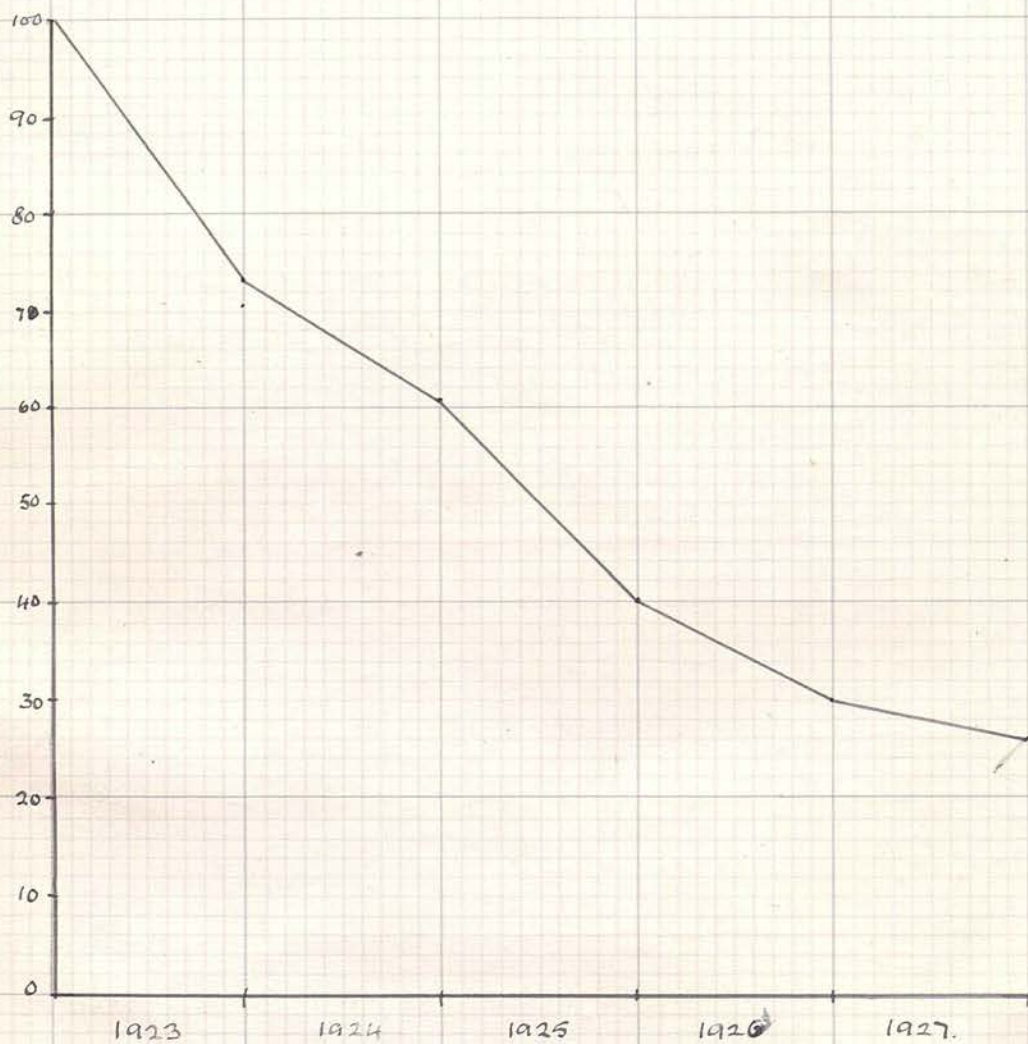
Similar figures were obtained by Lee¹² and Parmenter¹³ among Harvard University freshmen.

Leube³ reported the condition in 16% of 119 healthy soldiers, while Capitan¹⁴ found it in 40% of healthy recruits.

Bashford¹⁵ in any examination of 500 males of ages 14-30, found it in 41 cases (8%).

Most of the other figures are those of public school boys in their teens. Clement Dukes⁴ found it in 16% of new boys at Rugby College. Armstrong¹⁶ gives his incidence at Wellington College as 15% and Moxom¹⁶ as 10%.

The disparity in the various figures may be due to differences in technique and it is probable that observers finding constantly high percentages are using very delicate methods, and that with the ordinary technique, /



Graph showing percentage of cases who continued to
pass protein during re-examination 1923-27

technique, their figures would be much lower.

In the present series, of the 1250 cases examined, protein was discovered in the single specimen of urine passed at the first examination, in 107 cases (8.5%). Of these, 5 cases (Nos. 9, 18, 21, 39, 91) were considered on further examination (by the accessory renal tests referred to, taken in conjunction with the physical signs and the history) to be either organic renal disease or borderline cases and were not considered with the group under investigation.

The incidence of benign proteinuria, i.e. un-associated with any signs of organic kidney disease, was thus reduced to 102 cases (8%). Of these it has been possible to follow for the full 5 year period, 98.

STATISTICAL RESULT OF URINARY RE-EXAMINATION.

Repeated re-examination of the proteinuria patients during the 5 year period of observation gave results as follows. (The figures refer to the percentage positive on one or more examinations during that particular year).

End of 1st Year	-	73%
" " 2nd "	-	61%
" " 3rd "	-	40%
" " 4th "	-	32%
" " 5th "	-	26%

The figures show that on re-examination a rather definite and constant number of the cases continued to/

to show protein in the analysis of a single specimen of urine but that as time went on the percentage diminished gradually until at the last examination only 26% of the original number showed proteinuria.

During the re-examination the individuals making up the positives varied largely, especially in the earlier year periods. Thus some individuals who had shown protein previously would continue to show it. Others who had it at the last examination would have none, while others previously negative would prove positive.

AGE PERIOD DISTRIBUTION.

At the commencement of the observations 5 years ago, the age incidence of the cases was as shown in the table.

Age Period Incidence at the Beginning.

Age-Period	Incidence.	Percentage of total Albuminurics (for that year).
15-17 Years	38 cases	37.2
18-20	35 "	34.4
21-25	29 "	28.4

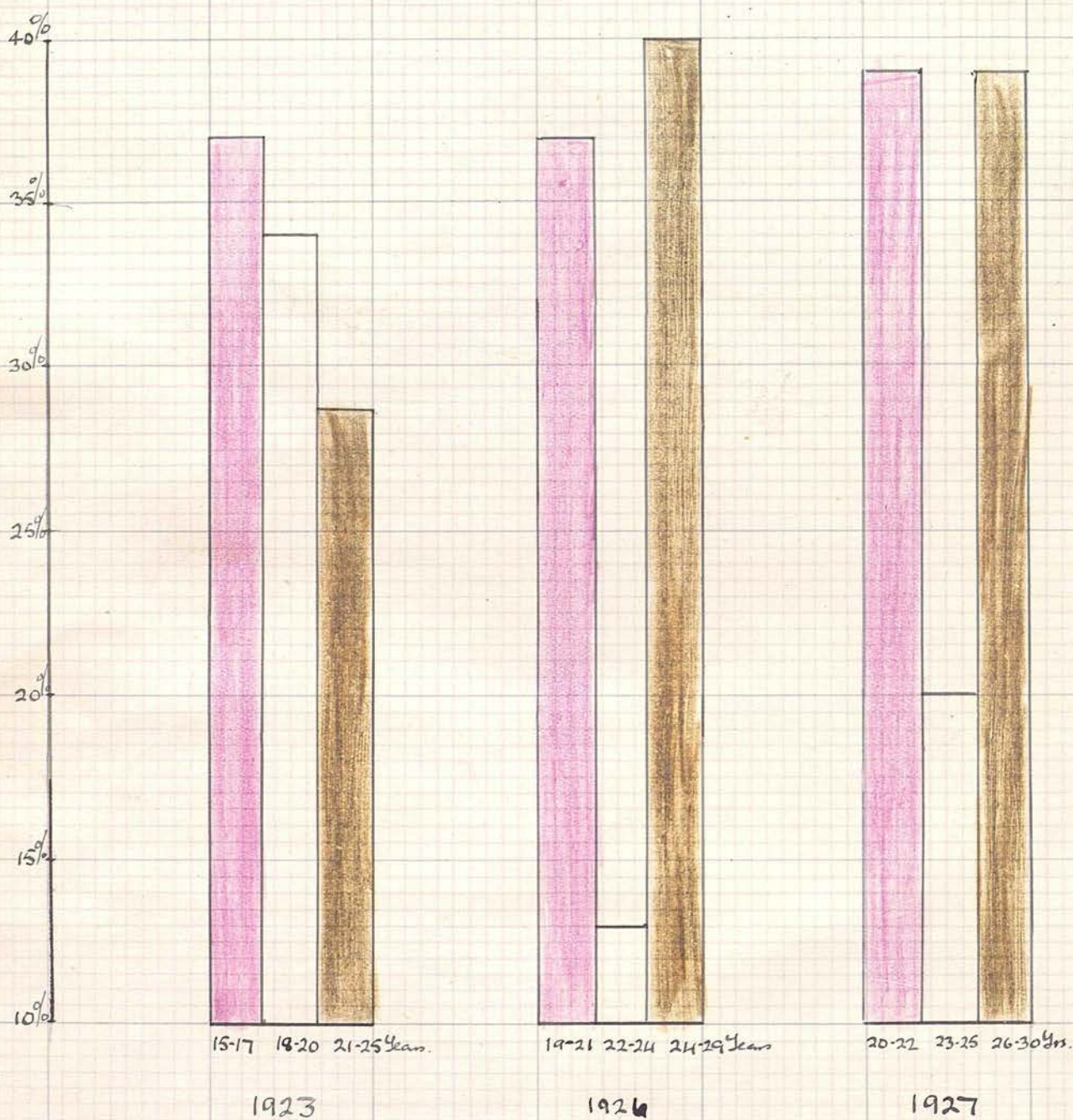


Diagram illustrating age-period distribution of Proteinurias
at commencement and end of observation period.

Age Period Incidence at end of 4th year.

Age Period.	Incidence.	Percentage of total Albuminurics (for that year).
19-22 Years	12 cases	37
22-24 "	7 "	23
25-29 "	13 "	40

Age Incidence at end of 5th year.

Age Period.	Incidence.	Percentage of total Albuminurics (for that year).
20-22 Years	10 cases	39
23-25 "	6 "	22
26-30 "	10 "	39

At the end of the 4th year of observation it was noticeable that although there was a steady disappearance in the incidence of proteinuria, in certain cases it showed evidence of persistence into age periods at which the condition is usually considered to be not very frequent. Each case showing the condition was re-examined systematically and in no instance could any alteration be detected in the physical condition to suggest transition into organic renal disease.

Such persistence of the proteinuria into the later age periods suggested a routine urine examination/

examination of male patients at the continued age-periods (30 upwards).

In the following 12 months, opportunities of such examination of 106 cases between the ages of 30-50 years were available. In 5 cases protein was discovered on more than one occasion, which on further investigation was apparently unassociated with an organic renal condition.

The details of these cases are as follows:-

CASE 1. aged 38. Gas-metre inspector. Came for advice re. ingrowing toe-nail. Previous health good. Has never been absent from work for 3 years from sickness. No history of previous infectious disease.

Afternoon specimen of urine passed in surgery contained albumin (1% Esbach) and no globulin. No casts or cells. No cardiac hypertrophy. B.P. 140. No oedema. Arteries soft. No headaches 90 or eye symptoms. No changes in fundus oculi. Concentrates urea at 2%. $\frac{N}{D} \frac{1}{3}$. Morning specimen of urine passed immediately after rising contained no protein. Within the next 12 months 5 further urinary tests were done. 2 contained protein. The patient is still in good health.

CASE 2. Motor-engineer, aged 42. Came for dressing for cut hand. Previous health good except for Influenzal Pneumonia in 1918. No other infectious diseases.

Afternoon specimen contained small amount of albumin. No globulin. No casts or cells. No cardiac enlargement. B.P. 150 95. No oedema. No arterial thickening. No headaches or eye-symptoms. Fundus oculi normal. Early morning sample of urine passed on arising - no protein.

Urine subsequently examined 7 times, protein being found twice. This patient is still well and says he feels very fit.

CASE 3. Salesman, aged 36. Came because of muscular rheumatism. Appendicectomy 12 years ago. Health record otherwise good. No history of any infectious diseases.

Morning specimen (11 a.m.) of urine contained definite albuminuric cloud. No globulin. A few hyaline casts. No cells. Heart - not enlarged. B.P. $\frac{135}{90}$. No oedema. No headache or visual disturbances. Fundus oculi normal.

5 subsequent urinary examinations; one showed moderate amount of albumin; others free. Early morning sample after rising, free. Concentrates urea at 2.4%.

This patient is still well.

CASE 4. Motor-driver. Aged 42. Came because of deafness. Previous health good except for "muscular rheumatism". No history of previous infectious disease.

Evening specimen contained albumin and some globulin. A few hyaline casts. No cells. Urea concentration 2.2%. No cardiac enlargement. B.P. $\frac{150}{90}$. No oedema. Arteries not thickened.

Early morning specimen after rising - no protein.

Urine subsequently examined on 6 occasions and protein was found twice.

Fundus oculi was normal. No headaches or visual disturbance.

Patient still feels very well.

CASE 5. Motor mechanic. Aged 34. Came because of "Indigestion". Previous health good except for dyspeptic symptoms. Had perforated duodenal ulcer 6 years ago. No history of previous infectious disease.

Evening specimen contained albumin in moderate amount ($\frac{1}{4}\%$ Esbach). No casts or cells. No cardiac hypertrophy. B.P. $\frac{135}{90}$. Arterial walls not thickened. No oedema.

Urine subsequently examined on 8 occasions, on two of which protein was found. The early morning specimen after rising was always protein free.

The patient still feels very well and strong, apart from the gastric symptoms.

The studies of the age distribution of the cases of proteinuria are of numbers too small, and the period of observation in the 5 above cases too short from which to draw definite conclusions but suggest

- (1) that benign albuminuria may not be confined to youths and young men but that it may be a definite entity at later age periods.
- (2) that the real explanation of certain middle-aged albuminurias may be the presistence of a functional proteinuria from youth.

TYPES OF PROTEIN IN THE BENIGN PROTEINURIAS.

In recent British literature "proteinuria" is rapidly displacing "albuminuria" in reference to the condition under discussion, owing to the adoption of the word by Mackenzie Wallis and to his belief that albumin is not the only or even the main protein present in the urine of these cases.

In a paper read before the Royal Society of Medicine in May 1920, Wallis¹⁸ states that proteinuria may comprise 2 proteins (1) serum albumin, and (2) serum globulin or euglobulin, which, according to his own work and that of Langstein¹⁹ is the protein which precipitates out when acetic acid is added to the cold urine.

In organic renal disease the main protein is albumin (in the proportion of albumin 6 : globulin 1), while in the so-called "functional" proteinurias, the globulin is in relative excess.

He divides non-nephritic proteinuria into 3 classes (1) Functional, adolescent or postural, in which during intermittent periods unrelated to known causes, protein is passed, consisting of both albumin and globulin (albumin 2 : globulin 1). (2) A condition known as "Leaky Kidney" defined as a permanent, long-standing, profuse proteinuria, really an excessive globulinuria/

globulinuria (albumin 1: globulin 2), showing no renal or structural change or evidence of organic or progressive disease, but tending to remain. (3) Physiological albuminuria following exercise in which the protein present is largely serum albumin.

As early as 1886, Jaccoud¹⁹ and Maguire²⁰ had reported globulin in these cases, confirmed by Teisser²¹ but its presence had been largely ignored.

Saito²² found that the body precipitated by acetic acid varied absolutely and relatively in the same person and Harrison²³ found no constant proportion of globulin, the variation being from 0 to 100 percent in the same person at different times.

Hamil and Blackfan²⁴ examined the urine of 124 presumably healthy children of ages up to 14 years and albumin was found in the urine of 110 of the children examined, or 88%. It was present in 2 forms (1) serum albumin (2) an albuminous body which they termed "nucleo-protein"; this was precipitated by acetic acid in the cold, and is apparently the same as the "globulin" of Wallis. The proportions of the different proteins present in the 124 children were as follows: 34 or 27.4% showed serum albumin, alone 4, and in combination 30; 106 or 85.4% "nucleo-protein". "Nucleo-protein" was therefore present 3 times as often as serum albumin. The authors state their belief that a greater number of observations would have/

have demonstrated, at some time or another, the presence of "nucleo-protein" in the urine of every child.

On the other hand Maclean's work¹¹ on the British soldiers does not agree with these findings. He examined 60,000 troops and he tested all the urines that showed a fair amount of protein, with acetic acid and he found that in only 2-3 cases was there any precipitate at all. Eventually, he gave up the acetic acid test, because he concluded that the very small number of cases which gave any reaction showed that even if a positive reaction was of any consequence, there might be many cases in which there was no acetic acid reaction at all, which were undoubtedly cases of functional proteinuria.

The work of Mackenzie Wallis and Langstein²⁵ on the identification of the proteins has been further amplified by Welker²⁶ whose researches disclose that the chemical tests for the identification of the types of protein are difficult of interpretation but that the precipitin tests (with ammonium sulphate) accurately identify most of them.

By means of the precipitin test he found that the protein in urine may be identified as

- (1) Coagulable protein not precipitated in the cold by acetic acid (serum albumin, pseudo-globulin).
- (2) Coagulable protein precipitated in the cold by acetic acid (euglobulin).

(3)/

- (3) Non-coagulable protein precipitated in the cold by acetic acid (mucin and nucleo-protein).
- (4) Non-coagulable protein not precipitated by acetic acid at all (proteoses).

There was no simple test for separating euglobulin, mucin and nucleo-protein available.

As the investigators referred to above (Wallis, Hamil and Blackfan, etc.) used only the chemical test and not the precipitin test, they, of course, included mucin and nucleo-protein as well as euglobulin in what they termed "globulin".

TECHNIQUE OF TESTS EMPLOYED IN PRESENT OBSERVATIONS.

A simple test was used throughout.

(1) Albumin. A test-tube was filled about two-thirds full with clear urine. The upper portion was heated to boiling point in the flame and from 5-6 drops of 33% acetic acid added. The contents of the tube were viewed against a black back-ground, and those urines were considered normal which exhibited no cloudiness.

(2) In testing for the "globulin bodies" (viz. euglobin, mucin and nucleo-protein) - which will hereafter be collectively referred to simply as globulin, a few drops of the 33% acetic acid were added to the unheated urine. The test was considered positive if precipitate came at once.

The results of the observations into the type of protein present at the first examination as determined by the heat and cold acetic acid tests were as follows:-

Results of observations on type of Protein present.

Total number of cases examined	1250	
Total number showing benign proteinuria	102	
Total number giving a positive protein test on the addition of acetic acid to the cold urine	26	25.5%
Total number giving a positive protein test on addition of acetic acid to the heated urine	100	98%
Total number more positive in the heated than in the cold urine	92	90%
Total number more positive in the cold than in the heated urine	4	4%
Total number positive in the cold only	2	2%
Total number positive in the hot only	73	71.5%

These results show that 25.5% of the urines showing a positive protein reaction contained either euglobulin, nucleo-protein, mucin or all of them, which/

which precipitate out when acetic acid is added to the cold urine and that in 2%, they alone were present. As 90% of the total were more positive in the hot urine, it is reasonable to assume, that at least in this number, serum albumin or pseudo-globulin, or both, were present and that in at least 71.5% they alone were present, since this percentage reacted only in the heated urine.

INFLUENCE OF AGE UPON TYPE OF PROTEIN PRESENT.

Globulin was much more prevalent in the patients at the younger ages; thus, of the 26 cases showing "globulinuria", 20 were between the ages of 15 and 20 years. Re-examination of these cases now, at the end of 5 years, when the patients are in the early twenties, showed the persistence of it in only 2 cases (2 brothers, showing probably the condition of "leaky kidneys", Cases No. 24 and 25).

One concludes from these results that globulin (eu-globulin, nucleo-protein, and mucin) is not a prominent component of the urinary protein in the benign proteinurias.

AMOUNT OF PROTEIN PRESENT.

In general the quantity passed was small, in some cases merely a haze. The amount varied in each individual/

individual case from time to time. The largest amount registered by the Esbach albuminometer was 1%.

Those urines which showed the largest amount of albumin were those in which globulin was found.

OTHER URINARY CHARACTERISTICS IN BENIGN PROTEINURIA.

I. Oxalate crystals. The association of oxalate crystals with albumin in these cases has been noticed by several observers. Maclean noted it in a very large number of the urines in his soldiers in France. Bashford did not notice any such frequent association in his cases amongst the boys and men in the post-office service.

In the present series of cases, microscopical examination of the centrifugalised deposit in the sample passed at the first examination showed oxalate crystals in 31 cases (30%).

II. Casts. Casts were present in 3 cases and were of the hyaline variety. On two subsequent examinations of these 3 cases, casts were not found.

III. Specific Gravity. There was no definite association between the height of the specific gravity and the presence of protein. The readings varied from 1000-1020.

IV. /

IV. Cells. No cells were found except in case No. 53 after the cycle-race on the track in whom a few red blood cells were found. These were absent at a subsequent examination.

THE ORTHOSTATIC REACTION IN BENIGN PROTEINURIA.

This is understood to designate a condition in which the urine excreted while lying down is protein free, while that passed after rising is more or less albuminous.

Saito²² states that 90% of his positive protein cases in 148 healthy school-boys were of the orthostatic type.

Bashford¹⁵ in examination of 1000 boys of 14 years of age at the General Post Office found albumin to be absent from the morning or after-rest specimen in 40 out of 43 who showed benign albuminuria. And in 41 benign cases out of 500 males between the ages of 14-30, albumin was found to be absent from 30, in the morning or after-rest specimen.

Hamil and Blackfan²⁴ however definitely imply that their examination of 124 children, ages up to 14 years, showed that most of their positive cases were not orthostatic because the amount of protein was not increased by the upright posture or exercise.

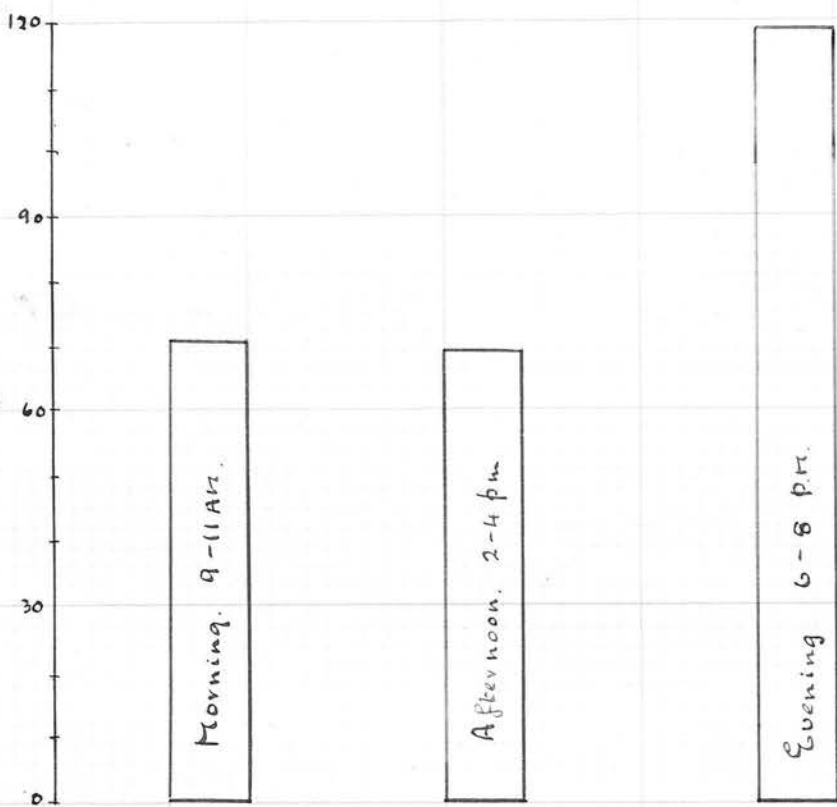
Recent observations by Russell²⁷ seem to show that the orthostatic reaction is not confined to benign proteinurias, as many patients suffering from scarlet fever, who presented no albumin during the period/

period of rest in bed, developed orthostatic albuminuria after getting up, which in some cases was permanent and associated with increasing blood-pressure, and which he regarded as definite nephritis. He states that postural influence shows itself in many and possibly all forms of albuminuria, and certainly in the "parenchymatous" varieties of nephritis, acute and chronic. He concludes that he regards the complete cessation of albumin excretion in the horizontal position, characteristic of "orthostatic albuminuria" merely as the extreme example of the influence of posture.

In the present series of cases the urine of all the 102 cases, passed immediately after rising in the morning, was protein free. 23 of the patients were engineers who were at intervals engaged on night "shifts" and again in each case the urine passed on getting up in the evening after rising from rest during the day was free of protein.

It was possible to test the orthostatic reaction in 22 cases who, during a re-examination, passed protein in a sample passed at the surgery in the morning. Each case was asked to return home and to lie in bed for 6 hours, at the end of which time, to pass a sample and bring it for examination. In 20 of the cases, the protein had disappeared.

These /



Graph illustrating day-period
incidence of Proteinuria.

These results show that orthostatism has a very definite effect in the production of protein in the type of case under consideration.

CYCLICAL VARIATIONS OF ORTHOSTATIC ALBUMINURIA.

Pavy²⁸ described the passage of the albumin in orthostatic or functional cases as occurring in a cycle and named the condition "cyclical albuminuria". Each case on first rising in the morning was free from protein but with the assumption of the upright posture, albumin shortly began to escape from the kidney, especially after breakfast, owing to lack of adaptation of the vaso-motor mechanism in these cases. As the day went on, such adaptation gradually occurred and the albumin disappeared, so that the evening specimen was protein free.

In the periodic urinary re-examination of the present cases, note was taken in the positive results, of the period of the day in which the examination was done. The results are shown in the following table. (The re-examinations were distributed fairly uniformly over the 3 periods of the day.)

Morning(9-11 a.m)	Afternoon(2-4 p.m)	Evening(6-8 p.m.)
74	72	118

These results show that a cycle of the nature described by Pavy is not the rule.

PHYSICAL CONSTITUTION AND HABITUS OF THE CASES
OF YOUTHFUL BENIGN PROTEINURIA.

Most of the observers of proteinuria in youths in public schools describe a physical condition of these patients as so constant that the condition can often be forecast merely from the physical appearance of the patient. (Clement Dukes, Kingston Fox, Goodhart, Armstrong, West, Post and Thomas).

The typical picture is described by them as an anaemic "weedy" youth, thin, rapidly growing, of the frail-lanky type; the chest long and narrow; the scapulae and abdomen prominent with lumbar lordosis and the heart apparently hypertrophied and overacting; with widespread evidence of vaso-motor instability and a tendency to faintings ("chapel-fainters"); with pale skin, moist, cold, cyanotic hands and dilated pupils, subjective lassitude, headaches, palpitations, vertigo, sensitiveness to cold and altogether evidence that the subject is a highly-strung, neurotic individual.

In adults Teisser²¹ noted a tendency to urticaria, chilblains, purpura, eczema, and various erythemas.

More recent investigators especially in America, especially Lee and Parmenter^{12 & 13} have laid stress on the presence of defects in body posture, finding the frequent association of proteinuria with poor posture. /

posture. (This is really a refinement of the previous observations on lordosis (orthostatism)).

On the other hand, Bashford¹⁵ from an investigation of 500 cases between the ages of 14 and 30 and Norman³⁵ from his observations of 1787 boys between the ages of 7 and 15 years, state that they found no definite physical type showing the conditions.

The results of the present observations do not in general agree with those of the public school medical officers quoted. There was a history of faints in only 4 cases. Only 10 could be described as of the neurotic, asthenic type with poor posture and a certain degree of lordosis. The others were strong non-emotional youths of average muscular development, with good complexions, some rapidly growing, or over-grown, others not. One noticed no tendency to skin reactions.

In general it appeared that proteinuria in the ordinary civil population is not associated with any particular type of youth or man, with a so-called "nervous disposition", or with lordosis.

THE INFLUENCE OF MUSCULAR EXERTION
UPON PROTEINURIA.

The influence of exercise has been studied by W.C. Collier²⁹ in England in the urines of boat-crews, and by Dunhill and Patterson³⁰ in Australia. Albumin was invariably present after the races.

Such post-exercise albuminuria has since been found by several workers to be associated with changes in the pulse pressure. D.R. Hooker³¹ found in his laboratory that immediately after exercise the diastolic and systolic pressure were much increased, the systolic more than the diastolic so that the pulse pressure was increased, this condition giving way at once to a fall of both pressures, accompanied by a decrease of pulse pressure. Faint traces of albumin were found in the first urines voided after the exercise. The albumin increased in amount with the post-exercise fall of pulse pressure regardless of the actual systolic and diastolic pressures and disappeared with the return of the pulse pressure to normal.

Barach, Boyce and Savage³² determined the systolic and diastolic pressures in a number of Marathon runners before and after the races. The urines were normal before they ran. After the exertion the urine contained casts, blood and albumin and this change was /

was accompanied by a marked diminution in the amplitude of the pulse pressure.

A. Of the present series of cases it was possible to make exercise observations in 14 patients who were members of amateur football teams. A urinary sample was examined before the match, also two post-exercise specimens, one passed immediately after the match and another about 2 hours later in the evening. Pulse pressure readings were not taken.

The results are as follows:-

(All these cases had shown protein in the urine on one or more occasions during the year the observations were made).

o	means no protein
a	" albumin
g	" globulin
+	" protein in moderate amount
++	" " " large amount.

TABLE /

Table showing effect of exercise (football or rugby)
on proteinuric cases.

	Before game.	Immediately after.	2 hours after.
Case I (No.17)	o	o	o
" II (No.45)	o	o	o
" III (No.36)	Trace a.	+ a.	+ + a
" IV (No.29)	o	o	o
" V (No.32)	o	trace a.	o
" VI (No.56)	Trace a.	o	Trace a.
" VII (No.15)	Trace g.	o	o
" VIII (No.58)	o	o	o
" IX (No.41)	Trace g.	o	Trace g.
" X (No.52)	o	o	o
" XI (No.95)	o	+ (a)	Trace a.
" XII (No.66)	o	o	o
" XIII (No.75)	Trace a.	o	o
" XIV (No.92)	o	o	o

B. More strenuous muscular exertion.

Two cases of the present series (Nos. 5 and 53) were scratch men in public track-cycle racing competitions and the opportunity was taken of making some observations/

observations both on pulse pressure and urinalysis before and after a sprint of 5 miles in one case and 1 mile in the other. (the first race in which each competed).

Case No. 5.

I. Before the race. (1 mile)

Blood pressure readings. $\frac{135}{95}$. Urine free from protein. (Protein had been found apart from exercise a few weeks previously).

II. After the race. (5 minutes after finish).

Pulse readings. $\frac{140}{95}$ mm.

Urine - no albumin present. Cold acetic acid test - negative. Microscopically - no casts, no cells.

2 hours later:- Pulse pressure $\frac{135}{95}$.

Urine - albumin in moderate amount.

Acetic acid test - slightly positive. Microscopically - a few hyaline casts. No cells. Following day - no protein.

Case No. 53.

I. Before the race (5 miles).

Blood-pressure $\frac{130}{90}$ mm.

Urine - no protein.

II. After the race (5 minutes after finish).

Blood-Pressure $\frac{140}{100}$ mm.

Urine - protein present in moderate amount.

Cold acetic acid test negative. Microscopically
- no casts, no cells.

2 hours later:- Blood-pressure $\frac{135}{95}$ mm.

Urine - large amount of albumin. Acetic acid
test - slightly positive. Microscopically, -
hyaline casts and a few red blood corpuscles.

24 hours later, urine was protein free.

One may conclude from these results that in cases
of benign proteinuria, violent muscular exertion has
marked influence in inducing the proteinuria while
ordinary, moderately strenuous games have little or
no influence.

THE INFLUENCE OF FEBRILE INFECTIONS
UPON THE PROTEINURIA.

It has long been noted that a large number of varieties of febrile infections in healthy people may be attended by a slight trace of albuminuria without accompanying clinical symptoms or signs that can be rightly ascribed to renal change.

Thus Rand³³ states that this febrile albuminuria is so common that it may accompany any febrile attack which produces so slight a change in the glomeruli of the kidney as a cloudy swelling; that the amount of albumin is always slight, and that it disappears from the urine with the cessation of the fever. He explains it by saying that in subjects of acute pyrexia, albumin passes into the urine because the process of filtration by which the urinary secretion is affected at the glomeruli is, by reason of the abnormally elevated temperature, conducted under entirely abnormal conditions. The effect of the elevated temperature upon the product of filtration is to cause a greater relaxation of the walls of the blood vessels, thus causing them to yield to the hydrostatic pressure of the blood streaming through them. This alteration in the condition of filtration at the glomeruli will vary according to the individual. One person may endure a high elevation of the temperature for several weeks/

weeks without any such changes, while another after a short duration will excrete albuminous urine.

During the period of observation of the present series of cases, opportunities were afforded of watching the effects on the urinary protein produced by the development of some acute febrile conditions. The results of such pyrexias on a certain number of the control cases were also noted. In order to have similar diseases for purposes of comparison, attention was confined to the two commonest minor febrile complaints met with in ordinary practice, viz. acute naso-pharyngeal infections (febrile) and acute follicular tonsillitis.

Of these, there were opportunities of examining 46 cases amongst the proteinurias and 35 amongst the controls.

I. The Proteinuric Group.

No. of cases tested	46
No. who developed protein in the urine during the febrile period (recumbent)	34
Protein present.	
(1) Albumin alone or chiefly	14
(2) Globulin alone or chiefly	20.
Protein absent.	12
Number showing continuations of Proteinuria after 2 days cessation of fever (ambulatory)	6

In all cases the proteinuria had ceased by 4th day after drop of temperature.

II. The Control Cases (see Appendix).

No. of cases observed	35
No. who developed protein in the urine during the febrile period (recumbent).	29
Protein present.	
(1) Albumin alone or chiefly.	20
(2) Globulin alone or chiefly.	9
Protein absent.	6
Number showing continuation of proteinuria after 2 days cessation of fever.	3

In all cases the proteinuria had ceased by the 4th day after fall of temperature.

One concludes from these results:-

- (1) that cases of benign proteinuria, as compared with non-proteinurias, show no predisposition to "febrile albuminuria".
- (2) that when present, there is a tendency to the predominance of globulin over albumin.
- (3) that the passage of the protein does not tend to continue after the cessation of the pyrexia.

THE PREDISPOSITION OF PROTEINURICS TO NEPHRITIS IN DISEASES IN WHICH NEPHRITIS IS A FREQUENT COMPLICATION.

If adolescent functional albuminuria were due to actual renal injury, it would predispose to nephritis and consequently it would be expected that in such albuminuria, nephritis, as a primary condition or as a complication of diseases in which kidney inflammation is frequent, would be of common occurrence.

Maclean's observations¹¹ on this point are of great value. Albuminuria had been found in 5% of 60,000 healthy soldiers examined, yet of all the cases in which organic disease of the kidney arose, none or scarcely any came from the number of those in whom temporary albuminuria had been noted. "Previous albuminuria" Maclean concluded "plays little or no part in the etiology of war nephritis".

In the present cases, there was no instance of acute nephritis as a primary disease during the period of observation, nor did any case show, from physical signs in blood-pressure or cardiac enlargement, at the end of the period, any suggestion of organic renal disease developing.

There was, during the period, one instance in which there was opportunity for observation of the influence of attack by disease in which nephritis is a/
a/

a frequent complication. This was Case No. 63, who in the 3rd year of the period during which the observations had been carried out, developed an attack of scarlet fever. This patient, then aged 23, had on 3 separate occasions shown protein in moderate amount, the last occasion being 2 months prior to the onset of the scarlet fever. The case, which was a moderately severe one, with temperature ranging to 102° , was nursed at home and the urine was examined daily for 5 weeks from the onset.

The details of the case are as follows:-

May 3rd 1924. Onset of scarlet fever, with typical tongue, rash and throat. T. 102.4 . 24 hour urinary specimen - no protein present.

May 4th. T. 101° - no protein.

" 5th. T. 101.4 - no protein.

" 6th. T. 100 - slight trace of albumin.

No casts. No oedema.

" 7th. T. 99.4 - no protein.

" 8th. T. 98.4 . - no protein.

The temperature remained normal from this date onwards and on no occasion was any protein found.

May 28. Patient allowed to get up. Peeling in progress. A small haze of albumin in the afternoon specimen, but no casts or cells, and no oedema.

May 28 - June 4. Patient was up but confined to his room. No protein found.

June 5 - 12. Patient getting about outside. No protein found.

After/

After this date the patient went away for a holiday and there was no opportunity of examining his urine until his return on July 2nd, on which date a sample passed in the afternoon was free from protein.

From that date to November 1927 ($3\frac{1}{2}$ years later) the urine has been examined on 14 different occasions and only on one occasion, Sept. 2nd 1926 when he was suffering from an acute coryza, was any protein found (trace albumin, no casts). In November 1927 blood-pressure readings were $\frac{130}{90}$ and there was no cardiac enlargement.

These results suggest that cases of functional albuminuria show no predisposition to nephritis with the development of an intercurrent attack of a disease in which nephritis is a frequent complication.

ASSOCIATED SPHYGMOMANOMETRY OF THE BENIGN PROTEINURIAS.

Many of the pioneer workers made observations on this point from clinical examination without the aid of the present instruments, whose facilities were not in use at that time.

Clement Dukes⁴ believed that he was able to determine increased arterial tension in all his cases at Rugby School.

Craig³⁴ on the other hand suggested that albuminuria was present as the result of low arterial tension and stated that he was able to make albumin disappear by raising the blood pressure.

Later workers, using the modern instruments have presented their findings with numerical data.

Norman³⁵ states that in his cases no increase in blood pressure could be ascertained (no figures given).

Lee¹² amongst the 250 benign proteinurias of the Harvard University freshmen (ages 16-24) found wide variations in blood pressure amongst the various cases, with Systolic readings of over 140 mm. in 12.8%. He concluded that alterations in blood-pressure in these cases were inconstant and associated with the nervous disturbance attendant on the examination, that is, that it represented types of "labile" blood-pressure. But he found that the incidence of proteinuria/

proteinuria was twice as great in the group with such "labile blood pressure", as in the total group.

Parmenter¹³ in an examination of 513 Harvard freshmen found systolic blood pressures varying from 120-140 mm. in the 25 cases showing albumin, and of these 2 had varying degrees of hypertension and tachycardia, therefore an unstable circulatory condition.

Estimation of the pressures in the present cases was done with an instrument of the mercurial variety by the auscultatory method. The pressure recorded at the first examination was repeated at a later examination so as to correct any influence exerted at the initial testing by emotional upset. The readings were taken first in the recumbent posture and then in the standing up position. Finally the readings were taken in all the cases who continued to show urinary protein, at the end of the 5th year.

The exact figures are given under the case-sheet of each individual case. The highest pressure (systolic) recorded was 140 mm. and the lowest 110, the diastolic varying from 75 mm. to 95 mm. These figures are within the range of what is usually considered normal pressures.

The final readings in those cases showing proteinuria in the 5th year, were not perceptibly increased/

increased (allowing for a slight rise due to increased age).

POSTURAL INFLUENCE ON BLOOD-PRESSURE READINGS.

This question was first gone into by Erlanger and Hooker³⁶. They found that when the normal individual changes from the horizontal to the upward position there is a rise in systolic blood pressure, accompanied by an equal rise in diastolic, so that the pulse-pressure remains practically constant. Whereas in orthostatic albuminurics, on such change there is a fall in pulse pressure owing to a diastolic rise, the systolic remaining practically constant.

Bass and Wessler³⁷ repeated these observations but failed to find any constant indication of blood pressure changes.

In the present cases, the results are given under the individual case sheets, the readings being made on the day during which albumin was being passed. 82 showed a rise in pulse pressure on changing from the horizontal to the upright position, 8 showed a drop, while 12 showed no alteration. From which it is concluded that the postural changes in blood pressure in the benign albuminurics are not specific and approximate to those recorded in the normal individual.

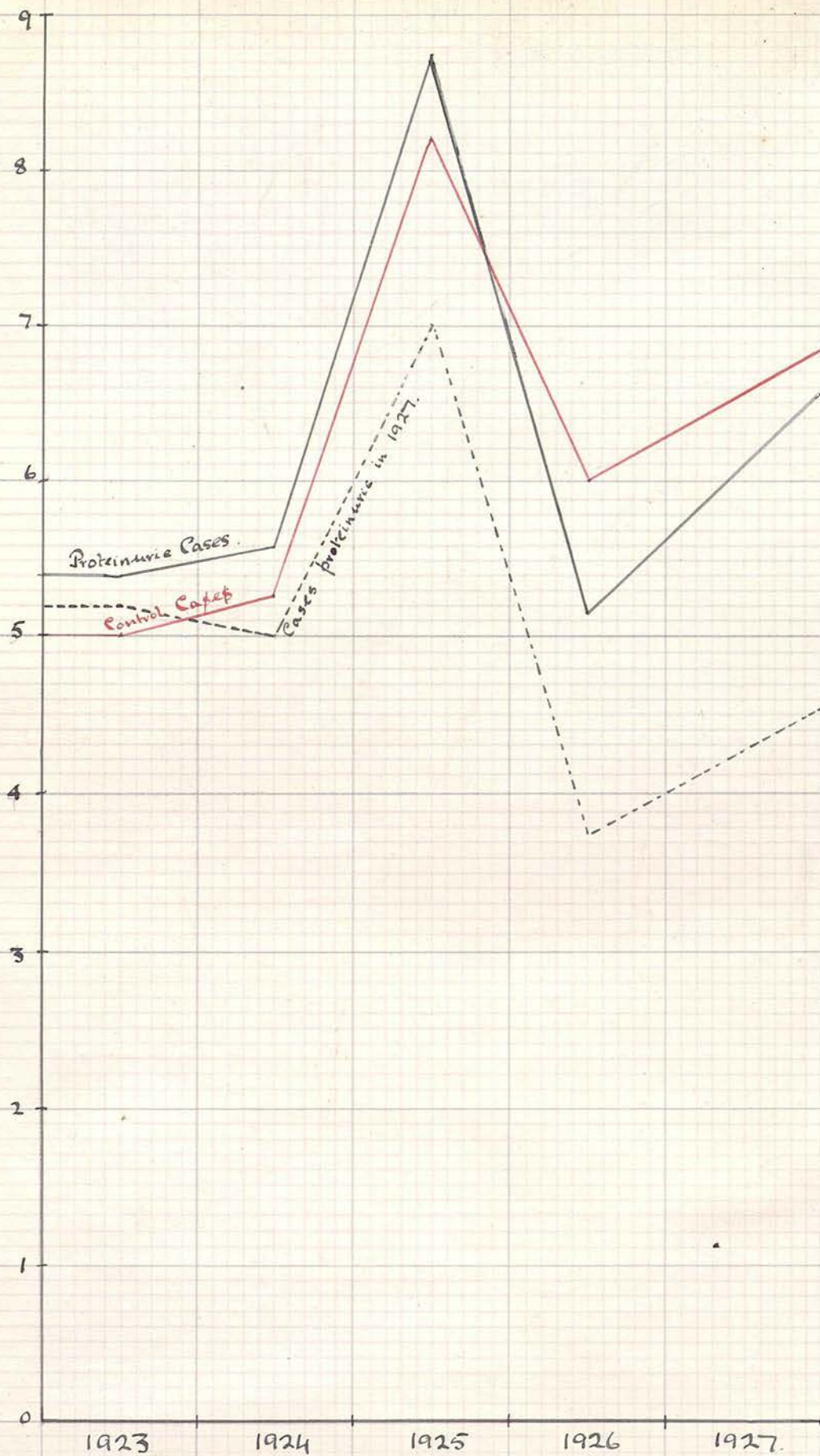
RELATION TO PHYSICAL EFFICIENCY AND
SICKNESS RECORD (MORBIDITY RATE).

This side of the question viz. the observing of the effect, if any, of this functional youthful proteinuria upon general physical efficiency and the sick record of the individual was the main object of the present investigation and one upon which few observations have been recorded.

It has been possible to keep 98 of the 102 cases under continuous observation during the full period.

In order to have a standard with which to compare, sickness record notes have been kept during the period of an equal number of controls, consisting of unselected male patients, of approximately the same ages, same social and working conditions and exposed to the same sickness hazards, who at the routine urinary examinations of males at the commencement of the investigation, showed no proteinuria.

Of the 102 positive cases, one has been killed by a motor cycle accident; one died following operation for appendicitis and two left the city during the period and no details of their later health are available. The remaining 98 are all alive and in good health. 26 still show the occasional passage of protein. None of these show any evidence of organic disease.



Proteinuric Cases
Control Cases
Cases who continued to pass protein 1923-1927.

The details of the health records are given with the individual case sheets.

The following table summarises the records.

Sickness record in average number of days absent from work per annum. (surgical cases excluded).

(See graph opposite)

Year	Controls	Proteinuric cases.	Cases still showing Proteinuria in 1927.
1923	5.07	5.42	5.19
1924	5.27	5.56	5.03
1925	8.19	8.75	7.03
1926	6.02	5.13	3.76
1927	6.84	6.66	4.53

The spring of 1925 was the occasion of an influenza outbreak in the city. The rate of sickness was increased but was nearly equal in the two classes. The figures for the other years do not show any marked divergency.

Qualitative Morbidity Data.

The following table summarises the results of the analysis of the disease incidence. Surgical conditions are excluded.

The figures refer to the total number of cases of each disease, and only to those which were of sufficient severity to necessitate absence from work.

Disease Incidence.

Disease.	Control cases.	Proteinuric Cases.
Acute Upper Respiratory Catarrhs. (Coryza group)	146	123
Influenza	89	95
Follicular Tonsillitis	43	47
Dyspepsia.	25	25
Lower Respiratory Catarrhs. Bronchial group.	23	30
Acute Gastro-Enteritis	19	17
Rheumatic and Myalgic Conditions.	12	8
Furunculosis	8	7
Catarrhal Jaundice	3	2
Neuralgia and Neuritis	0	4
Lobar Pneumonia.	1	0
Scarlet Fever	0	1
Unclassified	1	3

The figures show that the disease incidence is not any higher in the proteinuric cases and that there is no proclivity to any special disease or type of disease.

C O N C L U S I O N S .

- I. That Benign Proteinuria occurs in about 8% of healthy young males between the ages of 15-25 years in ordinary civil conditions.
- II. That its presence is intermittent and varies with unknown factors.
- III. That the condition is associated with no particular type of man or youth, nor with a so-called "nervous disposition", nor with oxaluria, nor with any abnormal functioning of the cardiovascular system.
- IV. That the urinary protein is chiefly albumin, but that in a considerable proportion of cases in the younger age-periods, the acetic-acid bodies (globulin, mucin and nucleo-protein) are also present.
- V. That the condition is not influenced by ordinary muscular activity.
- VI. That it does not predispose to nephritis either as a sequela or as a complication of intercurrent diseases.
- VII. /

- VII. That the condition tends to disappear but that in many cases it may persist as a benign condition to a later period in life than usually thought.
- VIII. That it has no influence on physical efficiency or morbidity rate and that there is no proclivity to any special group of diseases associated with it.
-

REFERENCES.

1. Ultzmann: Wien: med. Presse 11: 81: 1870.
2. Bright, Richard: Guy's Hospital Rep. 1827.
3. Leube: Virchow's Arch. f. path. Anat. 72:145: 1878.
4. Clement Dukes. Brit. M.J. 1905. ii. 848-500.
5. Grainger Stewart and Gulland. Proceed. Roy. Soc. Edin. 1887.
6. Fox, R.H. Brit. M.J. 1911. i. 1113.
7. Saundby, R. Glasgow Med.J. 1884: XXI. n.s.413-420.
8. Goodhart, J.F. Proc. Roy. Soc. Med. 1910-11. IV. Med. Sect. 109.
9. Dublin, L.I. Am. J. Hygiene. 1: 301: 1921 and Vital statistics in relation to life insurance. N.Y. 1916.
10. Barrenger and Warren. Arch. Int. Med. June 1912.
11. Maclean, H. Gt. Britain. Med. Research Comm. Spec. Regt. Series 43.
12. Lee, R.I. Med. Clin. North Am. 3: 1059. Jan. 1920.
13. Parmenter, D.C. Boston Med. & S.J. 183: 677: Dec. 9, 1920.
14. Capitan: Thesis. Paris, 1883.
15. Bashford, H.H. Lancet, 1926. II. 1305-1307.
16. /

16. Armstrong. Med. Soc. Trans. 1905.
17. Moxon. Guy's Hosp. Reports 1878.
18. Wallis, Mackenzie. Proc. Roy. Soc. Med. 13 Sect. Med. 96: 1920.
19. Jaccond: Clin. med. de la Pitre 1887.
20. Maguire: Lancet 1: 1106: 1886.
21. Teisser: Semaine méd. 19: 425: 1899.
22. Saito, H. Am. J. Dis. Child. 22: 388. Oct. 1921.
23. Harrison, G.A. Lancet 2: 991: Nov. 1921.
24. Hamil, S.M. and Blackfan, K.D.: Am. J. Dis. Child. I: 139: Feb. 1911.
25. Langstein, L. Albuminur. 1907.
26. Welker, W.H. quoted by Calvin et al. J.A.M.A. Vol. 86, p.1821.
27. Russell, J.W. Lancet. Vol.II. p.683. 1925.
28. Pavy: Med. Soc. Trans. Vol.XXVII, p.95. 1905.
29. Collier, W. Brit. Med. J. 1907. I, p.4.
30. Dunhill and Patterson. Intercolon. M.J. Austral. 1902, p.334.
31. Hooker, D.R.: Johns Hopkins Hosp. Rep. 1904. 12. 346-378.
32. Barach, Boyce and Savage: Am. Phy. & Educ. Rev. Dec. 1910. Jan. Feb. March, Apr. & May 1911.
33. Rand, H.F.: Mod. Med. & Bact. Rev. Oct. 1899.8.244.

34. Craig: Brit. Med. Jour. 1886. I. p.333.
35. Norman, R.H.: Clin. Jour. No. 1618. p.330.
36. Erlanger and Hooker. Johns Hopkins Rep. 12:
145. 1904.
37. Bass, M.H. and Wessler, H.: Arch. Int. Med. 13:
39. Jan. 1914.
-

A P P E N D I X.

Sickness record over period 1923-1927 of 98 youths and young men, ages 15-25 years (at the commencement) whose urine did not contain protein. These were unselected individuals who were examined at the same time as the proteinurics for the purpose of ascertaining the incidence of proteinuria in the young male population. The health record of these "controls" has been used as a standard against that of the proteinuric cases in the determination of the influence of the proteinuria in the latter upon physical efficiency and sickness record.

Only those ailments which necessitated absence from work were recorded. Surgical conditions were excluded.

S.R. ... means sickness record, the numeral following being the number of days absent during that year.

CASE 1. Aged 20. S.R. 1923. 0. 1924 4 days Coryza.
1925 3 days Coryza. 1926 3 days Coryza.
1927. 3 days Influenza.

CASE 2. aged 15. S.R. 1923 3 days Coryza.
1924. 0. 1925 17 days Influenza. 1926 0.
1927. 7 days Coryza.

CASE 3. aged 19. S.R. 1923 0. 1924 7 days Bron-
chitis. 3 days Coryza. 1925 0.
1926. 5 days Coryza. 1927 0.

CASE 4. /

- CASE 4.** aged 24. S.R. 1923 14 days Influenza.
1924. 3 days Coryza. 4 days Dyspepsia.
1925. 4 days Coryza. 1926. 0.
1927. 15 days Bronchitis.
- CASE 5.** aged 16. S.R. 1923 4 days Dyspepsia.
10 days Tonsillitis. 1920 0. 1925 0.
1926 0. 1927 0.
- CASE 6.** aged 18. S.R. 1923 0. 1924 7 days Influenza.
6 days Coryza (feverish) during which
passed albumin and globulin in small amounts,
former predominating. 2 days after cessation of
fever, protein free. 1925. S.R. 4 days Coryza.
1926 0. 1927. 10 days Influenza.
- CASE 7.** aged 19. 1923 7 days Coryza. 1924 0.
1925 0. 1926 7 days Influenza. 3 days
Coryza. 1927. 3 days Coryza. 4 days
Tonsillitis.
- CASE 8.** aged 23. S.R. 1923 0. 1924 9 days Bronchitis.
1925 4 days Coryza. 1926 0.
1927 4 days Coryza. 11 days Influenza.
- CASE 9.** aged 17. S.R. 1923 9 days Gastro-Enteritis.
1924 10 days Influenza. 1925 0.
1926 10 days Influenza. 1927 0.
- CASE 10.** aged 25. S.R. 1923 0. 1924 0. 1925 15
days Dyspepsia. 1926 0. 1927 10 days
Influenza.
- CASE 11.** aged 16. S.R. 1923 14 days Influenza.
1924 6 days Tonsillitis. During febrile
period passed albumin and small trace
globulin. After 2nd day of fall of temperature,
protein free. 1925 4 days Gastro-Enteritis.
1926 3 days Coryza. 5 days Coryza. 1927 12 days
Febricula.

CASE 12. aged 25. S.R. 1923 15 days Dyspepsia.
1924 0. 1925 0. 1926 7 days Coryza.
1927 0.

CASE 13. aged 21. S.R. 1923 0. 1924 5 days
Feverish Coryza during which passed small
amount of albumin and a small trace of
globulin. 3 days later (afebrile) protein free.
1925 17 days Influenza. 7 days Tonsillitis.
1926 3 days Coryza. 9 days Influenza. 1927 10 days
Influenza.

CASE 14. aged 17. S.R. 1923. 3 days Coryza.
1924 10 days Influenza. 1925 0. 1926 0.
1927 3 days Coryza. 9 days Influenza.

CASE 15. aged 20. S.R. 1923 0. 1924 0. 1925. 4
days Coryza. 1926 15 days Tonsillitis.
1927 0.

CASE 16. aged 15. S.R. 1923 5 days Rheumatism.
1924 10 days Influenza. 9 days Bronchitis.
1925 0. 1926 0. 1927 15 days Tonsillitis;
during febrile period passed albumin in moderate
amount and a small trace of globulin. 3 days after
cessation of fever, still passed a small trace of a.
(No g.) 2 days later - no protein.

CASE 17. aged 21. S.R. 1923 3 days Coryza. 4 days
Coryza. 1924 4 days Coryza. 10 days
Influenza. 1925 17 days Influenza. 7 days
Tonsillitis. 1926 16 days Influenza.
1927 0.

CASE 18. aged 16. S.R. 1923 0. 1924 7 days Influenza.
1925 0. 1926 21 days Catarrhal
Jaundice. 1927 9 days Influenza.

CASE 19. aged 22. S.R. 1923 14 days Bronchitis.
1924 0. 1925 0. 1926 4 days Coryza.
1927 0.

- CASE 20. aged 25. S.R. 1923 0. 1924 7 days
Influenza. 1925 0. 1926 0.
1927 6 days Gastro-Enteritis.
- CASE 21. aged 16. S.R. 1923 7 days Coryza. 7 days
Dyspepsia. 1924 3 days Coryza. 1925
3 days Coryza. 1926 7 days Gastro-
Enteritis. 1927 4 days Coryza.
- CASE 22. aged 23. S.R. 1923. 5 days Enteritis.
1924. 0. 1925 0. 1926. 60 days Acute
Rheumatic Fever. 1927 0.
- CASE 23. aged 18. S.R. 1923 0. 1924 5 days
Myalgia. 1925. 3 days Coryza. 4 days
febrile Coryza; during febrile period
passed small amount of globulin and a haze only of
albumin. After 2nd day of fall of temperature, protein
free. 1926 0. 1927 16 days Dyspepsia.
- CASE 24. aged 19. S.R. 1923 0. 1924. 14 days
Influenza. 1925 0. 1926. 6 days Furuncu-
losis. 1927. 0.
- CASE 25. aged 20. S.R. 1923. 14 days Influenza.
1924. 0. 1925. 17 days Influenza.
1926 10 days Influenza. 1927 10 days
Influenza.
- CASE 26. aged 24. S.R. 1923. 0. 1924. 12 days
Bronchitis. 1925 0. 1926. 10 days Bron-
chitis. 1927 0.
- CASE 27. aged 18. S.R. 1923. 13 days Bronchitis.
3 days Coryza. 1924. 11 days Influenza.
5 days Myalgia. 1925. 0. 1926. 0.
1927. 3 days Coryza; during febrile period,
urine contained no protein.
- CASE 28. /

- CASE 28.** aged 21. 1923 0. 1924 0. 1925. 10 days Influenza. 5 days Myalgia. 1926. 3 days Coryza. 1927. 0.
- CASE 29.** aged 15. 1923. 5 days Tonsillitis. 1924. 0. 1925. 0. 1926. 0. 1927. 9 days Dyspepsia.
- CASE 30.** aged 17. 1923 0. 1924. 12 days Influenza. 1925. 0. 1926. 3 days Coryza. 1927. 3 days Coryza. 5 days Dyspepsia.
- CASE 31.** aged 22. S.R. 1923. 4 days Coryza. 10 days Dyspepsia. 1924 0. 1925. 21 days Influenza. 1926. 0. 1927. 0.
- CASE 32.** aged 22. S.R. 1923 0. 1924. 7 days Coryza (two cases). 1925. 0. 1926. 5 days Tonsillitis; during the febrile period passed small amount of albumin and a trace of globulin. 2 days after drop of temperature, protein free. 8 days Myalgia. 1927. 14 days Influenza. 3 days Coryza.
- CASE 33.** aged 16. S.R. 1923. 4 days Coryza. 8 days Tonsillitis. During febrile period passed no protein. 1924. 0. 1925. 0. 1926. 0. 1927. 0.
- CASE 34.** aged 19. S.R. 1923. 21 days Acute Otitis Media. 1924. 0. 1925. 14 days, 3 separate attacks of Coryza. 1926. 0. 1927. 0.
- CASE 35.** aged 22. S.R. 1923. 8 days, 2 attacks of Coryza. 1924. 9 days Bronchitis. 1925. 17 days Influenza. 1926. 5 days Enteritis. 1927. 10 days Tonsillitis.
- CASE 36.** aged 15. S.R. 1923. 0. 1924. 4 days, 2 attacks of Coryza. 1925. 0. 1926. 0. 1927. 3 days Coryza. 10 days Acute Tonsillitis/

Tonsillitis, during the febrile period passed small amount of albumin and a haze of globulin. 3rd day after fall of temperature, protein free.

CASE 37. aged 21. S.R. 1923. 14 days Tonsillitis. During the febrile period, passed small amount of albumin and a haze only of globulin. Day after fall of temperature, protein free. 1924. 0. 1925. 3 days Coryza. 18 days Influenza. 1926. 0. 1927. 0.

CASE 38. aged 16. S.R. 1923. 0. 1924. 4 days Coryza. 7 days Tonsillitis. 1925. 0. 1926. 5 days Coryza. 1927. 14 days Bronchitis.

CASE 39. aged 24. S.R. 1923. 8 days, Furunculosis. 1924. 0. 1925. 7 days Influenza. 20 days Bronchitis. 1926. 0. 1927. 0.

CASE 40. aged 17. S.R. 1923. 5 days Coryza. 5 days Coryza. 1924. 14 days Bronchitis. 1925. 7 days Influenza. 14 days Quinsy. 1926. 4 days Coryza. 5 days Coryza. 4 days Coryza. 1927. 10 days Influenza.

CASE 41. aged 16. S.R. 1923. 0. 1924. 10 days Bronchitis. 1925. 21 days Influenza. 1926. 14 days Bronchitis. 1927. 48 days Lobar Pneumonia.

CASE 42. aged 23. S.R. 1923. 5 days Dyspepsia. 1924. 0. 1925. 0. 1926. 0. 1927. 3 days Coryza, 17 days Influenza.

CASE 43. aged 16. S.R. 1923. 0. 1924. 3 days Coryza. 7 days, Influenza. 1925. 7 days Influenza. 15 days, Tonsillitis; during the febrile period passed small amount of albumin and a trace of globulin. On 3rd day after fall of temperature, protein free. 1926. 10 days, Influenza. 1927. 0.

- CASE 44.** aged 23. S.R. 1923. 14 days, Influenza. 1924. 0. 1925. 5 days Gastro-Enteritis. 1926. 15 days Dyspepsia. 1927. 0.
- CASE 45.** aged 18. S.R. 1923. 0. 1924. 4 days Influenza. 1925. 0. 1926. 10 days Influenza. 1927. 6 days, Coryza.
- CASE 46.** aged 20. S.R. 1923. 3 days, Coryza. 1924. 7 days feverish Coryza, during which passed small amount of globulin and a trace of albumin. Day following fall of temperature, still passed a trace of albumin (no globulin). 2 days later, protein free. 1925. 14 days, Influenza. 5 days Tonsillitis. 1926. 0. 1927. 0.
- CASE 47.** aged 15. S.R. 1923. 5 days Enteritis. 1924. 0. 1925. 0. 1926. 3 days Coryza. 1927. 10 days Influenza.
- CASE 48.** aged 19. S.R. 1923. 14 days, Influenza. 1924. 6 days Enteritis. 1925. 7 days Influenza. 1926. 5 days Enteritis. 1927. 18 days Tonsillitis. During the febrile period, passed no protein.
- CASE 49.** aged 15. S.R. 1923. 0. 1924. 14 days Catarrhal Jaundice. 1925. 4 days Coryza. 1926. 0. 1927. 0.
- CASE 50.** aged 20. S.R. 1923. 6 days, Enteritis. 1924. 0. 1925. 0. 1926. 10 days, Furunculosis. 1927. 4 days Coryza. 6 days, Acute Tonsillitis. During the febrile period passed small amount of albumin and a trace of globulin. On 3rd day after fall of temperature, still passed a trace of albumin. 2 days later, protein free.
- CASE 51.** aged 19. S.R. 1923. 14 days, Bronchitis. 1924. 4 days Influenza. 1925. 4 days Coryza. 17 days Influenza. 1926. 8 days Enteritis. 1927. 10 days Influenza.

CASE 52. aged 20. S.R. 1923. 0. 1924. 4 days
Tonsillitis. 1925. 0. 1926. 3 days, Coryza.
14 days, Acute Tonsillitis. During the
febrile period passed moderate amount of albumin and
a small trace of globulin. 3 days later, afebrile -
protein free. 1927. 8 days Influenza.

CASE 53. aged 17. S.R. 1923. 3 days, Coryza.
1924. 7 days Influenza. 1925. 14 days,
Catarrhal Jaundice. 1926. 0. 1927. 0.

CASE 54. aged 18. S.R. 1923. 10 days Influenza.
9 days Dyspepsia. 1924. 7 days Influenza.
1925. 27 days Influenza. 1926. 3 days
Coryza. 7 days feverish Coryza, during
which passed small amount of albumin and a trace of
globulin. 2 days later (afebrile), protein free.
1927. 9 days Influenza.

CASE 55. aged 15. S.R. 1923. 5 days Coryza.
1924. 7 days Coryza. 1925. 5 days Coryza.
1926. 8 days Coryza. 1927. 4 days Coryza.
17 days Influenza.

CASE 56. aged 25. S.R. 1923. 14 days Tonsillitis.
1924. 4 days Coryza. 1925. 0. 1926. 0.
1927. 5 days Enteritis.

CASE 57. aged 20. S.R. 1923. 0. 1924. 0. 1925
3 days Coryza (febrile) during which passed
globulin and a small trace of albumin.
3 days later, (afebrile) - no protein. 17 days,
Influenza. 1926. 9 days Myalgia. 1927. 0.

CASE 58. aged 18. S.R. 1923. 14 days Furunculosis.
1924. 3 days Coryza. 5 days Dyspepsia.
4 days Coryza. 1925. 7 days Influenza.
14 days Quinsy. 1926. 3 days Coryza. 1927. A day's
Tonsillitis. During the febrile period passed a small
amount of albumin and a trace of globulin. 2 days
later (afebrile) - no protein.

CASE 59. /

- CASE 59.** aged 20. S.R. 1923. 14 days Influenza.
1924. 13 days Bronchitis. 1925. 0.
1926. 10 days Tonsillitis. During the
febrile period passed small amount of albumin and a
trace only of globulin; 2 days later (afebrile) protein
free. 1927. 3 days Coryza. 4 days Dyspepsia.
- CASE 60.** aged 15. S.R. 1923. 13 days Bronchitis.
1924. 0. 1925. 3 days Coryza. 20 days
Influenza. 1926. 0. 1927. 0.
- CASE 61.** aged 18. S.R. 1923. 0. 1924. 10 days
Influenza. 1925. 0. 1926. 9 days Enteritis.
1927. 8 days Enteritis.
- CASE 62.** aged 17. S.R. 1923. 3 days Coryza.
1924. 0. 1925. 3 days Coryza. 3 days
feverish Coryza. During the febrile period
passed globulin and a haze of albumin, which were
both absent on 2nd day after fall of temperature.
1926. 10 days Influenza. 1927. 0.
- CASE 63.** aged 25. S.R. 1923. 10 days Furunculosis.
1924. 14 days Furunculosis. 1925. 5 days
Coryza. 7 days acute tonsillitis.
1926. 0. 1927. 0.
- CASE 64.** aged 16. S.R. 1923. 7 days acute tonsil-
litis. During febrile period passed
albumin in excess of globulin. 2 days
later, afebrile, - protein free. 1924. 3 days Coryza.
1925. 0. 1926. 8 days Tonsillitis. 1927. 21 days
Influenza.
- CASE 65.** aged 19. S.R. 1923. 5 days Coryza.
1924. 0. 1925. 8 days Myalgia. 1926. 3 days
Coryza. 1927. 0.
- CASE 66.** aged 20. S.R. 1923. 0. 1924. 5 days Coryza.
1925. 4 days Coryza. 1926. 10 days Bron-
chitis. 1927. 10 days Influenza.
- CASE 67. /**

- CASE 67. aged 23. S.R. 1923. 0. 1924. 0. 1925. 0
1926. 7 days Coryza. 1927. 10 days Quinsy.
- CASE 68. aged 15. S.R. 1923. 4 days Dyspepsia.
1924. 3 days Coryza. 10 days Influenza.
1925. 7 days Tonsillitis. During febrile
period, passed globulin and a trace of albumin.
3 days later (afebrile) - protein free. 1926. 5 days
Dyspepsia. 1927. 7 days Coryza.
- CASE 69. aged 18. S.R. 1923. 0. 1924. 0. 1925.
17 days Bronchitis. 1926. 0. 1927. 0.
- CASE 70. aged 20. S.R. 1923. 0. 1924. 6 days
feverish coryza, during which passed
albumin with a slight haze of globulin.
Both absent 2 days later, afebrile. 12 days Bronchitis.
1925. 0. 1926. 7 days Coryza. 1927. 17 days Dyspepsia.
- CASE 71. aged 18. S.R. 1923. 3 days Coryza. 7 days
Influenza. 1924. 7 days Influenza.
1925. 3 days Coryza. 1926. 4 days feverish
coryza, during which passed no protein.
1927. 0.
- CASE 72. aged 16. S.R. 1923. 0. 1924. 0. 1926.
10 days Influenza. 1927. 14 days feverish
coryza during which passed albumin in small
amount and a trace of globulin. 3 days later (afebrile)
still a little albumin. 2 days later, protein free.
- CASE 73. aged 25. S.R. 1923. 13 days Coryza.
1924. 7 days Dyspepsia. 1925. 5 days
Coryza. 1926. 0. 1927. 10 days Influenza.
- CASE 74. aged 20. S.R. 1923. 0. 1924. 5 days,
Gastro-Enteritis. 1925. 21 days Influenza.
1926. 10 days Influenza. 1927. 12 days
Tonsillitis.
- CASE 75. aged 17. S.R. 1923. 0. 1924. 0. 1925. 0.
1926. 5 days Coryza. 1927. 0.

- CASE 76. aged 19. S.R. 1923. 4 days Enteritis.
1924. 3 days Coryza. 7 days Dyspepsia.
1925. 5 days Coryza. 1926. 0. 1927. 5
days Coryza.
- CASE 77. aged 16. S.R. 1923. 0. 1924. 0. 1925. 0.
1926. 10 days Quinsy. 1927. 7 days Coryza.
- CASE 78. aged 23. S.R. 1923. 5 days Coryza. 7 days
Influenza. 1924. 3 days Coryza. 5 days
Dyspepsia. 1925. 28 days Influenza.
1926. 0. 1927. 0.
- CASE 79. aged 20. S.R. 1923. 0. 1924. 0. 1925.
27 days, Influenza. 1926. 3 days Coryza.
1927. 10 days acute tonsillitis; during
febrile period passed small amount of albumin and a
trace only of globulin. Absent 3 days later (afebrile).
- CASE 80. aged 18. S.R. 1923. 5 days feverish coryza,
during which passed globulin and small
amount of albumin. 3 days later (afebrile)
both absent. 1924. 8 days Quinsy. 1925. 0.
1926. 10 days Bronchitis. 1927. 0.
- CASE 81. aged 24. S.R. 1923. 5 days Enteritis.
1924. 7 days Influenza. 1925. 3 days
Coryza. 1926. 5 days Dyspepsia. 1927.
4 days Coryza; 10 days Bronchitis.
- CASE 82. aged 20. S.R. 1923. 0. 1924. 0.
1925. 5 days feverish coryza during which
urine contained no protein. 1926. 0.
1927. 15 days Coryza.
- CASE 83. aged 16. S.R. 1923. 0. 1924. 10 days
Influenza. 1925. 0. 1926. 10 days Influenza.
1927. 0.
- CASE 84. /

- CASE 84.** aged 19. S.R. 1923. 8 days Quinsy.
1924. 3 days Coryza. 1925. 15 days Influenza. 7 days Tonsillitis during which passed albumin in small amount and trace of globulin; 3 days later (afebrile), protein absent.
1926. 12 days Rheumatism. 1927. 10 days Tonsillitis.
- CASE 85.** aged 16. S.R. 1923. 0. 1924. 0. 1925. 5 days, Coryza; 9 days, Tonsillitis.
1926. 0. 1927. 0.
- CASE 86.** aged 20. S.R. 1923. 0. 1924. 10 days, Myalgia. 1925. 0. 1926. 7 days Coryza.
1927. 8 days Tonsillitis.
- CASE 87.** aged 23. S.R. 1923. 0. 1924. 0. 1925. 5 days Coryza. 1926. 5 days, feverish coryza, during which passed small amount of albumin and a trace of globulin. 2 days later (afebrile), protein absent. 3 days Coryza.
1927. 0.
- CASE 88.** aged 19. S.R. 1923. 3 days Coryza.
1924. 3 days Coryza. 1925. 3 days Coryza.
19 days Influenza. 1926. 10 days Myalgia.
1927. 7 days, feverish coryza during which urine contained no protein.
- CASE 89.** aged 25. S.R. 1923. 0. 1924. 5 days Furunculosis. 1925. 5 days Coryza. 5 days Enteritis. 3 days Coryza. 1926. 0.
1927. 8 days Rheumatism.
- CASE 90.** aged 15. S.R. 1923. 0. 1924. 0. 1925. 5 days Coryza. 1926. 7 days Dyspepsia.
1927. 21 days Influenza.
- CASE 91.** aged 19. S.R. 1923. 3 days Coryza.
1924. 5 days Coryza. 1925. 0. 1926. 0.
1927. 0.

CASE 92. aged 16. S.R. 1923. 0. 1924. 3 days Coryza; 5 days acute Tonsillitis; during febrile period of latter passed small amount of globulin and a haze of albumin. 3 days later (afebrile) - protein absent. 1925. 21 days Influenza. 1926. 3 days Coryza. 7 days Tonsillitis. 1927. 7 days Dyspepsia.

CASE 93. aged 20. S.R. 1923. 0. 1924. 0. 1925. 21 days Influenza. 1926. 7 days Dyspepsia. 1927. 0.

CASE 94. aged 15. S.R. 1923. 5 days, feverish coryza during which passed moderate amount of albumin and a trace of globulin. 2 days later (afebrile) - no protein. 3 days Coryza. 1924. 3 days Coryza. 1925. 4 days Coryza. 1926. 0. 1927. 10 days Dyspepsia.

CASE 95. aged 19. S.R. 1923. 0. 1924. 0. 1925. 0. 1926. 10 days Influenza. 1927. 0.

CASE 96. aged 18. S.R. 1923. 0. 1924. 5 days Coryza. 5 days Coryza. 1925. 7 days Influenza. 1926. 0. 1927. 17 days acute Tonsillitis. During febrile period passed small amount of albumin and a haze of globulin. 2 days later (afebrile) trace of globulin. 3 days later, protein absent.

CASE 97. aged 20. S.R. 1923. 0. 1924. 8 days Coryza. 6 days Coryza. 21 days Bronchitis. 1926. 7 days Coryza. 1927. 0.

CASE 98. aged 15. 1923. 8 days feverish coryza, during which passed moderate amount of albumin and a trace of globulin. 3 days later (afebrile) - protein absent. 1924. 0. 1925. 4 days Coryza. 16 days Influenza. 1926. 6 days Tonsillitis. 1927. 6 days Coryza.